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Cerebrocortical Necrosis in Cattle and Sheep*

BY

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SUMMARY.—The paper describes a nervous disease of cattle and sheep, characterised clinically by circling, progressive ataxia, decubitus, nystagmus, hyperaesthesia, tonic spasms, opisthotonos and coma usually ending in death. Flock and herd morbidity was low, mortality very high. Disseminated necrosis of the cerebral cortex was found in all the cases studied. Lesions in other parts of the brain were seen inconstantly. The clinicopathological picture had all the appearance of a distinct entity to which the name cerebrocortical necrosis has been given. Attempts to transmit the disease by experimental inoculation failed. Analysis of the cerebral lesions suggests that its pathological basis is anoxia, but the cause has not been determined.

Introduction

DURING the past 6 years a nervous disease of cattle and sheep, an apparently distinct clinicopathological entity, has been diagnosed in the Pathology Department at Weybridge and more recently given the name cerebrocortical necrosis (Terlecki & Markson, 1959). Until the spring of 1959 the disease appeared to occur only sporadically and usually as single cases. Since then several outbreaks have been encountered in various parts of England and Wales, but particularly in East Anglia. The purpose of this paper is to present a clinicopathological description of the disease, the results of some laboratory experiments and a discussion of the aetiological possibilities.

Incidence

In the 3 years 1957 to 1959, material was received from 54 sheep, sent from the Ministry's veterinary investigation centres at Aberystwyth, Bristol, Cambridge, Leeds, Liverpool, Newcastle, Norwich, Reading and Weybridge. Some of this came from single cases, but most of it—37 brains—came from East Anglia and represented 13 outbreaks which occurred in the early summer of 1959 (Spence, Stevens, Saunders & Harris, 1960). With the exception of 6 ewes, the oldest 2 years of age, the disease affected lambs aged from 2 to 7 months. Flock morbidity ranged from 1 to 6 per cent. without sex bias; the mortality rate was usually 100 per cent. Cases have occurred in several breeds and under widely varying systems of flock management.

From the beginning of 1958 to the end of April, 1960, the disease was diagnosed in 49 bovine cases referred to us from the investigation centres at Cambridge, Newcastle, Norwich, Reading, Sutton Bonington, Weybridge and Worcester. The age incidence ranged from 10 weeks (1 case) to 11 months (1 case), but the disease was most commonly seen in animals between 3 and 6 months of age. Morbidity varied from single cases to over 60 per cent. of affected groups. Cases occurred in the Aberdeen Angus, Ayrshire, Friesian and Hereford breeds and their crosses, without apparent sex bias and under different systems of management and feeding.

Symptoms

The first symptom in sheep appeared to be aimless wandering which, within hours, progressed to circling, accompanied by amaurosis. Simultaneously ataxia developed and rapidly became worse. Affected animals would stagger and sway, or stand precariously with their feet wide apart and the head drawn back in a series of convulsive extensor spasms during which nystagmus and trismus became apparent. Eventually they would fall over to lie with legs extended, kicking intermittently until apparently exhausted. Once recumbent, they usually remained so until they died. By this time hyperaesthesia had appeared, so that handling affected sheep would result in violent galloping movements ending in extensor spasm with opisthotonos and nystagmus. Gradually, all movements became weaker, the opisthotonos persistent and more severe, coma ensued and ultimately death. It was not always possible to obtain complete clinical data; but, as

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far as is known, only one affected sheep recovered, the rest dying after a usually afebrile illness which varied in duration from a few hours to several days, the longest period recorded being about 3 weeks.

This description of symptoms is composite and based on information supplied by colleagues as well as on cases seen by the authors. Individually there was a wide range of variation which could make the clinical diagnosis of a single case difficult. Sometimes the syndrome developed so quickly that affected sheep were first seen recumbent in the convulsive stage. Some cases were referred to us as suspected listeriosis, louping ill, tetanus or delayed swayback; but taken as a whole the syndrome was well defined and suggestive of a disease process affecting the cerebral cortex.

The early stages of wandering and circling were not seen in cattle, the syndrome beginning with a short period of dullness and ataxia, leading to collapse and convulsive struggling with threshing of the limbs and, sometimes, weaving of the head and neck. Hyperaesthesia, opisthotonos, amaurosis, nystagmus, trismus and tonic extensor spasms were all prominent symptoms as in sheep and death usually ensued in a state of coma. Most affected cattle died after an illness lasting usually for from 2 to 6 days, exceptionally about 24 hours. Five cases were reported as having recovered, but only 2 of these became available for confirmatory necropsy and inclusion in the series discussed here.

Morbid Anatomy

In the 54 affected sheep which died or were killed in various stages of the disease the only consistent gross lesions found were confined to the brains, 51 of which were visibly abnormal. Many of the cerebral gyri, particularly in the dorsal and dorsolateral regions, were swollen and slightly soft to the touch. On slicing the cerebrum irregular areas of yellowish discolouration were found scattered throughout the cortex of both hemispheres. In some cases these areas were small, well defined and predominantly in the frontal region; in others they were more extensive and affected the whole thickness of the gyral cortex. In some brains the discolouration was clearly visible on the uncut gyral surface. Although always bilateral, the distribution of the lesions was not strictly symmetrical. The discoloured areas were usually soft and friable and quite distinct in texture from the contiguous healthy tissue. In the cases of longer duration a distinct line of separation was evident between the cortical grey matter and the subjacent white matter. In 2 cases areas of slight discolouration and softening were seen in the ventrorostral part of the cerebellum.

The lamb which made a clinical recovery was killed for necropsy 6 weeks after the cessation of symptoms. Careful search of the brain revealed a few discrete, small, round, circumscribed areas of dirty yellowish discoloration and softening, situated at the surface of the cerebral cortex, most of them in the frontal region.

In affected cattle necrosis was absent or scanty at the frontal poles, increasing in extent towards the occipital poles. As it did so, focal necrosis usually became laminar until, in the occipital lobes, the lesion extended as a yellowish, or dirty cream coloured, almost unbroken layer over most of the cortical area and one third to a half of the cortical thickness in depth. In many cases the deeper, supramedullary cortical tissue was pale and translucent as if oedematous, an appearance which sometimes broke through to the surface, breaching the discoloured, superficial lamina and occasionally isolating necrotic foci. In 5 cases, including one examined 2 months after clinical recovery, gross necrosis was slight and essentially focal; in 3 cases it was not observed at all. In one calf which had partially recovered 2 weeks after the onset of symptoms a few small, subarachnoid cysts were seen in the cerebral cortex of dorsolateral occipital gyri.

Gross discolouration of cerebellar foci was seen in only 1 case. With this exception, gross lesions were always confined to the cerebral cortex.

Histopathology

Brains from all the ovine cases and spinal cords from 16 of them have been examined histologically after fixation in 10 per cent. neutral formol-saline and paraffin embedding.

Lesions are present in the cerebral cortex of all the brains and show a wide range, not only from case to case, but within the same brain, presumably related to their severity, stage and rate of development. Because of the complex inter-relationship of such factors, however, the correlation of the histopathological pictures with the duration and severity of the symptoms is not always clear.

In brains from sheep which died within 24 hours of the onset of symptoms, the cerebral lesions are fairly constant. Under a low magnification they appear as focal or laminar areas of spongy texture (microcavitation) involving part or all of the thickness of the cortical grey matter. Higher magnification reveals the presence of enlarged perivascular spaces, pronounced perineuronal vacuolation and a general looseness of the matrix, which probably represent a state of oedema. Most neurons in the centre of an affected area are shrunken and angular, sometimes triangular in outline with strongly acidophilic cytoplasm. Their nuclei, too, are shrunken and pyknotic with scarcely recognisable nucleoli. A few neurons, particularly near the periphery of the lesion, are swollen with rounded outline and finely vacuolated cytoplasm. Occasionally a few lymphocytes and monocytes are visible in perivascular spaces and small perivenular haemorrhages are not uncommon. Meningeal blood vessels, particularly over affected parts of the cortex, are frequently congested, while the meningeal tissues are moderately infiltrated with lymphocytes, monocytes and occasionally neutrophiles.

In specimens from animals which survived for two or more days, microcavitation is usually less severe and mainly confined to the periphery of lesions which are of two types. In one the affected area has an ill-defined border and appears to be highly vascular due to an actual or apparent increase

in the number of capillaries present. Many vascular endothelial cells are swollen and some show mitotic nuclei. Most of the neurons seen are still in the shrunken state; but some are now only ghost cells while others seem to have disappeared leaving only a hole. Marked infiltration by microgliacytes and, to a lesser extent, astrocytes gives some lesions a highly cellular appearance. The other type of lesion consists of uniformly dense areas which stain more intensely than the normal tissue from which they are clearly demarcated by a zone of variably vacuolated tissue. Neuronal changes in such lesions are similar to those already described, except that shrinkage seems to be less severe and swelling more common. Microglial and vascular activity are confined mostly to the periphery of the lesions. Lysis and removal of the necrotic tissue by phagocytes appear to commence most frequently in the submeningeal zone and are sometimes accompanied or followed by similar changes in the middle or deepest zones of the cortex. Cyst formation has not been seen in any of the brains studied. Where the reaction is severe, the subjacent white matter may be oedematous. In focal lesions the lytic process seems to begin at the edge of the necrotic tissue. In several brains some affected parts of the cortex are reduced to a loose mesh of astrocyte fibres, the interstices of which are filled with phagocytes. Shrunken neurons can still be seen in this obviously later stage of the lesion. Peripherally there is a zone of moderate astrocytosis and vascular proliferation.

In sections of cerebral cortex from 3 animals which had survived for from 5 to 10 days after first showing symptoms, several neurons in this peripheral zone contain a basophilic, finely granular, cytoplasmic deposit which extends into the dendrites. Stained by haematoxylin and eosin these neurons closely resemble the ferruginated neurons found at the periphery of old cerebral infarcts in man; but Perl's Prussion blue reaction has failed to demonstrate iron in the granules. Von Kossa's method impregnated them with silver, however, suggesting that they consist of calcium phosphate or carbonate.

In 30 of the brains pathological changes are visible in the cerebellum as well. Many Purkinje cells have disappeared and those which remain look either degenerated or necrotic, some being swollen and weakly and diffusely stained, others shrunken with eosinophilic cytoplasm and pyknotic nuclei. Moderate to severe rarefaction of the granular layer is also a feature.

Focal neuronal degeneration, microgliosis and vascular activity have been seen in the mesencephalon of two cases and the mid-thalamus and hippocampus of one. No pathological abnormality has been found in any of the sections of spinal cord.

In cerebral sections from the lamb which made a clinical recovery, lesions are few and discrete. Most of them take the form of focal or laminar astrocytosis, but a few consist of central necrosis with evidence of lysis and repair at the periphery. Several neurons in the vicinity of such lesions are calcified.

Generally speaking the lesions in the bovine brains are qualitatively similar, differing mainly in degree and disposition. In several cases stratification of the cerebral cortex into a more or less continuous supramedullary zone of microcavitation, covered by a superficial layer of tissue containing degenerate neurons in a fairly solid eosinophilic matrix without microcavitation, is fairly regular and apparently corresponds to the deep, translucent lamina seen in the gross specimen covered by the superficial yellowish tissue. Sometimes the deeper, porous tissue has broken through to the surface, isolating short laminae of the superficial necrosis. But in many brains the pattern is not as regular, microcavitation with necrosis, and necrosis by itself appearing at different levels in the cortex. The meningeal reaction is more intense than in the sheep, sometimes comprising massive infiltration with macrophages which swell the pia-arachnoid to many times its normal thickness, penetrate into the cortex in the perivascular spaces or by extension from the surface, and occasionally appear as isolated foci in the mid-cortex. Vasculitis in the form of capillary endothelial swelling is variably present and appears more prominently than in the sheep.

In cerebral sections from the calf which was killed 2 weeks after the first sign of disease, the acute reaction is intense. Macrophage infiltration of the submeningeal cortex is locally severe with, in some fields, separation of superficial from the deeper cortex. In a few fields the superficial, necrosed cortex has disintegrated with the formation of a small, intergyral cavity filled with tissue debris and divided in two by the intact, but sometimes inflamed meninges. This is the only case in both cattle and sheep in which such cavitation has been seen. The chronic, reparative phase is visible elsewhere in the same sections in the form of submeningeal or mid-zonal astrocytosis.

Similarly disposed astrocytosis and a little residual vasculitis are the only evidence of disease seen in the cerebrum of the calf which survived 2 months from the onset of symptoms.

Cerebellar lesions, similar to those described in the sheep and confined to the vermis, are visible in 8 of the 49 brains examined. Moderate microglial activity, satellitosis, neuronophagia and microcavitation are present in the thalamus of one brain, the hippocampus being similarly affected to a lesser degree. In one brain with moderate cerebrocortical necrosis the lesion is also to be seen in the caudate nucleus.

In none of the bovine brains examined have calcified neurons been seen, as described in the ovine cases. (See art plates facing page 32.)

Bacteriology

No significant bacterial isolates were obtained from the heart's blood, liver, spleen and brain of the many cases examined bacteriologically.

Biochemistry

Estimations of the calcium, magnesium, copper and lead content of samples of blood, liver and kidney, as appropriate, from many cases were made

by the Biochemistry Department, Weybridge, for the Investigation Officers concerned, without finding evidence of hypocalcaemia, hypomagnesaemia, hypocuprosis or lead poisoning.

Transmission Experiments

In attempts to transmit the disease, 14 three-to-four-months-old lambs were inoculated intracerebrally or intravenously with undiluted cerebrospinal fluid, citrated whole blood, or a suspension of brain from animals in which cerebrocortical necrosis had been diagnosed. Brain for inoculation was suspended in 10 per cent. guinea-pig serum-saline to a concentration of 10^{-1}. Two different samples were used, one from a clinically early case, the other from an advanced case, in an attempt to cover possible variation in the concentration of an infective agent in the brain during the course of the disease. The experimental animals were kept on a normal diet with ample water and were all clinically normal during the 3 weeks following inoculation. They were subsequently used in other experiments and their brains examined as they came to necropsy. No histological abnormality was found. Two of these animals were still alive and apparently healthy 10 months after inoculation.

Inoculum	*Number of animals*	*Route of inoculation*	*Dose*
Brain of early case	2	i/c	1 ml.
	2	i/v	2 ml.
Brain of advanced case	2	i/c	1 ml.
	2	i/v	2 ml.
Cerebrospinal fluid	2	i/c	1 ml.
	2	i/v	5 ml.
Blood	2	i/v	10 ml.

Two calves were inoculated intravenously with a 10 per cent. suspension in 10 per cent. guinea-pig serum-saline of diseased cerebrum from an affected calf and survived for several weeks in apparently perfect health.

Discussion

The essential pathological feature of the disease described in this paper is necrosis of the encephalic grey matter, a lesion always present and disseminated throughout the cerebral cortex, frequently present in the cerebellum and occasionally seen in other parts of the brain. There are several recent references to nervous diseases of cattle and sheep in which local necrosis of the brain and spinal cord is the principal pathological feature. Plowright (1954) and Innes and Plowright (1955) have described a disease of lambs in Kenya, in which paralysis was associated with symmetrical poliomyelomalacia. Bilaterally symmetrical malacia of the basal ganglia, internal capsule and anterior colliculi was a constant finding in a disease of New Zealand lambs described by Hartley (1956). A possibly identical disease has been reported in Britain by Barlow (1958) and Robertson and Wilson (1958) and has also been recorded at the Central Veterinary Laboratory.

Jensen and his colleagues (1956) have described a nervous disease of young sheep and calves, with focal necrosis of the cerebral cortex, which they called polioencephalomalacia. In clinically recovered animals, which constituted a large proportion of those diseased, they found cortical cysts enclosed by a zone of astrocytosis. Elliott (1958) has reported focal necrosis of the cerebral cortex in cattle and sheep comparable to polioencephalomalacia and made a detailed histological study of brains from four bovine cases. Mullins, Hartley and Salisbury (1958) reported an outbreak of amaurosis in New Zealand lambs, associated with necrotic lesions in the cerebral cortex. Since then Hartley and Kater (1959) described a nervous disease of lambs associated with widespread, cerebrocortical malacia, which they considered to be a severe form of the disease reported by Mullins and his colleagues and comparable to the American polioencephalomalacia.

The disease described in the present paper has many features in common with polioencephalomalacia in the U.S.A., notably the character and distribution of the lesions; but the symptoms are not identical and the mortality rate of the British disease is much higher. Perhaps because of this high mortality rate certain features of the recovery phase, in particular the formation of cortical cysts described by Jensen, Griner and Adams (*loc. cit.*), have rarely been observed in the British cases studied at Weybridge. Apart from this the differences are small and possibly of little significance. The British disease is, however, virtually indistinguishable from the severe form of the New Zealand entity.

In none of the diseases quoted has the cause been identified. In the U.S.A. selenium intoxication was at one time considered a possible cause, but the theory was later discarded. Hartley and Kater (*loc. cit.*) observed the occasional association of polioencephalomalacia with bacterial infection (unspecified) of the meninges, which suggested to them that a bacterial toxin might play a part in causing the disease. They also mentioned the coexistence of the disease with focal symmetrical encephalomalacia in one flock and, indeed, in two brains and, having postulated elsewhere (Hartley, *loc. cit.*) a relationship between the latter disease and clostridial enterotoxaemia, suggested that clostridial enterotoxins might be involved in the pathogenesis of polioencephalomalacia. Without denying such a possibility, it must be pointed out that this suggestion was partly based on an unproven assumption.

The cause of cerebrocortical necrosis in Britain remains unknown. There seems to be sufficient evidence for discounting the possibility of an infectious agent: many sporadic cases have been diagnosed; cultural and microscopical examination of the central nervous system from affected animals for the presence of micro-organisms has consistently yielded negative results; experimental attempts at transmission have failed. An analytical approach to the lesions, however, reveals certain features which permit discussion of some aetiological possibilities. Significant in this respect seems to be the predominance of certain types of neuronal degeneration which correspond morphologically and tinctorially to the

ischaemic group of Greenfield's (1959) classification. According to Meyer (1959) the presence of such types of neuronal degeneration is of considerable diagnostic importance as "they enjoy a degree of specificity to anoxia and ischaemia greater than any of the other degeneration types". Another point relevant to this argument is the presence of shrunken neurons containing basophilic, calcareous granules, around the periphery of advanced lesions. Although their significance remains obscure, their morphological resemblance to the ferruginated neurons described in old infarcts in man may be more than coincidental (bearing in mind the species difference in the deposition of calcium salts in necrotic cells).

Causes of encephalic anoxia in its widest sense—anoxic anoxia, ischaemia—are many. The list, headed by circulatory disorder, contains among others several poisons (*e.g.* carbon monoxide, carbon disulphide, potassium cyanide) which, although differing in their mode of action, lead ultimately to anoxia. In man accidental poisoning by these substances leads to anoxic lesions in the brain similar to those found in cerebrocortical necrosis as described in this paper. Similar lesions have also been reproduced experimentally in cats by ligation of both vertebral arteries (Gildea & Cobb, 1930), and in monkeys (Hurst, 1940) and dogs (Haymaker, Ginzler & Ferguson, 1952) by the administration of a single dose of cyanide compounds.

The histopathological picture of cerebrocortical necrosis bears some resemblance to that produced in pigs by experimental salt and urea poisoning (Done, Harding & Lloyd, 1959) in which water restriction was shown to be an essential pathogenic factor. In a few of the outbreaks or cases of cerebrocortical necrosis investigated, water deprivation was observed; but most of the herds and flocks involved were amply supplied with fresh water.

The investigation of pastures and supplementary food as possible sources of poison produced negative results.

Careful consideration has been given to the hypothesis put forward by Hartley and Kater (*loc. cit.*) of the possible rôle of *Clostridium welchii* type D toxin in the causation of the disease. Spence *et al.* (*loc. cit.*) demonstrated clostridial toxin lethal for mice in the intestinal contents of only three ovine cases and obtained a gel diffusion precipitin reaction against *Cl. welchii* type D antiserum in 32 of 48 samples; but there was no consistent quantitative correlation between the toxin and the severity of the clinicopathological picture. Furthermore, in most of the flocks investigated by them, the lambs had been vaccinated against pulpy kidney disease or given pulpy kidney immune serum. To sustain this hypothesis, one would therefore have to consider either the possible ineffectiveness of the prophylaxis, or the breakdown of the induced immunity occurring under special circumstances and leading to selective involvement of the brain. Terlecki and Thomson have administered repeated, sublethal doses of *Cl. welchii* type D *epsilon* toxin intravenously to lambs without causing cerebrocortical necrosis. Subsequent injection of these lambs with multiple lethal doses of the toxin neither killed them nor caused cerebrocortical necrosis.

In view of what has been said about the pathological anatomy of cerebrocortical necrosis, this name may appear slightly inaccurate or even misleading to some readers. In suggesting it, however, the following points have been taken into consideration:—

1. The only constant and the most characteristic lesions are those in the cerebral cortex; lesions in other parts of the brain, however frequent, may be absent.

2. The underlying process is essentially necrosis, of which malacia is only one feature.

3. As long as the cause of the disease remains unknown, the name cerebrocortical necrosis will serve to differentiate it from other forms of encephalomalacia in which the nature, distribution and possibly the causes of the lesions are different.

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Cerebrocortical Necrosis in Sheep and Cattle

The Clinical Syndrome*

BY

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SUMMARY.—(1) *A nervous condition involving lambs, ewes and calves associated with cerebrocortical necrosis is described.*

(2) *Twenty-one outbreaks in sheep and* 12 *outbreaks in calves were encountered in East Anglia between November,* 1958, *and July,* 1960.

(3) *A comparison is made with similar nervous diseases described in the literature.*

(4) *The exact cause remains obscure, but the epidemiology suggests that a toxin or toxins are involved.*

Introduction

THIS paper describes in greater detail the clinical features of a syndrome first reported in Great Britain by Terlecki and Markson in 1959. In East Anglia during the period February, 1958, to July, 1960, 21 outbreaks of cerebrocortical necrosis in sheep and 12 outbreaks in calves were investigated. Both sheep and cattle showed similar nervous signs and young animals were most frequently affected. The number of animals affected in each outbreak varied and recovery was very rare. The only constant macroscopical lesions were confined to the grey matter of the brain, in particular the cerebral cortex in which there were bilateral, but not necessarily symmetrical, areas of yellow discoloration and swelling of affected gyri.

The condition was similar in many respects to polioencephalomalacia described by Jensen, Griner and Adams (1956) in the U.S.A. and Hartley and Kater (1959) in New Zealand. A full description of the histopathology of the disease is given in a separate paper (Terlecki & Markson, 1961).

The Disease in Sheep

History

Twenty-one outbreaks of cerebrocortical necrosis in sheep on 19 different farms were investigated in East Anglia between April, 1959, and July, 1960. On only 2 farms was the disease seen in both years. In 1 of these outbreaks only 1 lamb was lost in each of these years, but in the other, losses were more severe, 17 lambs being affected in June and July, 1959, and 8 ewes and 7 lambs affected in June and July, 1960. In 2 of the remaining 19 outbreaks ewes only were affected, in another 2, ewes and lambs were affected, while the remainder (15) involved lambs only. The highest percentage loss of lambs occurred in a flock of 150 ewes where 17 of 250 (6 per cent.) lambs died and the highest percentage loss in ewes was 6 of 120 (5 per cent.). In these outbreaks an average of 2 per cent. of the lambs and 4 per cent. of the ewes were affected. Recovery was rare, only 3 lambs which showed typical signs survived. The breeds represented included Southdown, Clun and Suffolk, and crosses of the latter 2 breeds with Border Leicester and Scottish Half-bred ewes. The most commonly affected lambs were crosses between Suffolks and Half-breds, the predominant cross in the area.

Management

The disease was seen on farms with soil types varying from fenland peat to very light land with chalk subsoil, generally on temporary leys but occasionally on permanent pastures. There appeared to be nothing unusual in these pastures and they contained a variety of herbage plants including Italian and perennial ryegrass, cocksfoot, meadow fescue, red and white clover, lucerne and trefoil. Most of the flocks received supplementary concentrate food, though in some it was available to the lambs only, as a creep feed. The concentrates varied widely in composition, but crushed oats, barley, sugar-beet pulp, chopped mangolds and various proprietary compounds were most commonly used. In all but 3 flocks minerals were available and supplied in the form of proprietary additives or mineral licks. With the exception of one farm the water supply was adequate and was mostly from the mains direct or by water cart. The rainfall during the spring and early summer of 1959 and 1960 was low and it was exceptionally hot and dry. The pastures, however, were well managed and there was no shortage of grass.

Most of the sheep were in good condition when first seen and losses preceding the onset of this disease had been negligible. Prophylactic measures had been taken against pulpy kidney disease in all but 6 of the flocks and usually consisted of vaccination of the ewes just before lambing and vaccination of the lambs at 4 to 6 weeks of age. In 2 flocks lamb dysentery antiserum was given to the lambs at birth.

Clinical appearance

In the early stages both ewes and lambs were often

* This paper was received for publication October 6th, 1960.

found wandering aimlessly, sometimes in circles; in other cases they stood motionless. Within a few hours, however, the majority of cases were unable to stand and lay on their sides with the limbs extended and head retracted. Some lay for long periods without moving, others paddled continuously, but any sudden noise produced violent galloping movements and tonic spasms when nystagmus was marked. The rectal temperatures in the early stages were often elevated. With the exception of 3 lambs, all sheep which showed typical clinical signs ultimately died or were killed *in extremis,* although the period of illness varied from 12 hours to 21 days. The age incidence varied from lambs of 6 weeks of age to broken mouthed ewes of 7 years or more, but the great majority of cases were in lambs of 8 to 16 weeks of age.

On one farm during the period May 19th to June 25th, 1959, 18 lambs aged 2 to 3 months were affected, 17 of which died or were killed *in extremis;* 16 of these were examined *post mortem.* From investigation of this outbreak the following general pattern of clinical signs was observed. At first only inappetence and scouring were noticed, but within 3 days the lambs began to wander aimlessly about the pasture making no attempt to graze or suck. They seemed unable to find their dams and often bleated continuously. The head was held high and the ears drooped; a proportion of the lambs gave the impression of being blind although the pupils reacted normally to light. Usually within 12 hours they developed ataxia and began to circle in a drunken fashion, sometimes showing a high stepping action. Some stood with legs splayed as if to maintain balance and their heads were retracted in a series of convulsive movements during which time there was marked nystagmus; they then fell over and kicked or lay with legs extended and head retracted. A few lambs died during one of these spasms, others regained their feet with difficulty. The final stage of recumbency followed quickly (Fig. 1). Some lay quite still for long periods, others paddled continuously and any mild stimulation resulted in violent galloping movements during which time the eyes assumed a bright alert appearance. Respirations were greatly increased. These paroxysms terminated with tonic spasms and opisthotonos (Fig. 2). Movements became more feeble and opisthotonos more marked with the duration of the disease; some of the lambs groaned continuously as if in pain. The pedal reflex was usually evident until the final stages when there were often continuous weak paddling movements. The lambs invariably died in an extended position with marked opisthotonos within 1 to 15 days of first showing nervous signs.

The clinical signs in the ewes examined varied slightly. Two ewes from one outbreak were first found with their heads turned towards their flanks. Although the position of the head could be corrected manually it immediately returned to the flank on being released. These ewes also showed unilateral facial paralysis and unilateral blindness*. A ewe from another outbreak had a very stilted gait and with its neck arched and head drawn back its general appearance was reminiscent of a camel (Fig. 3); any sudden noise or movement caused excitement and ataxia. Other ewes showed similar signs to the lambs (Fig. 4), scouring being a common feature.

Rectal temperatures varied considerably, but were usually slightly raised, in the region of 104° to 105° F. The lowest temperature recorded was 99° F. in a comatose lamb and the highest was 110° F. during the excitable stage. Respiration and pulse rates were slightly increased.

Treatment

Treatment varied widely and included various antibiotics, calcium, magnesium and phosphorus injections and pulpy kidney antiserum, none of which seemed to alter the course of the disease. In the less acute cases parenteral glucose saline seemed to prolong life. One of the 3 lambs which recovered had been affected 4 days before being sent to the laboratory. It was apparently blind and stood only when assisted, the remainder of the time it was recumbent and showed the typical signs already described. Some degree of trismus was evident, but the swallowing reflex was present and by feeding milk and milk substitute by stomach tube for one week and force bottle feeding for a further 3 weeks it gradually regained its strength, became adapted to its disability and started to graze. Another lamb from the same flock recovered after being less seriously affected. Although they behaved normally, neither of these lambs thrived during the 6 weeks following their recovery.

Post-mortem *findings*

At the Cambridge Veterinary Investigation Centre 67 typical cases were examined *post-mortem,* 6 of these being ewes. The only constant lesions were found in the brain although epicardial haemorrhages, excess pericardial fluid and enlargement of the mesenteric lymph glands were relatively common. In some lambs the kidneys were soft as in enterotoxaemia and in a few the intestines were congested, but in only 3 was there marked enteritis. In 3 lambs killed in particularly good condition at an early stage extensive oedema of the mesentery and mesenteric lymph glands was a notable feature.

In all but 3 lambs and one ewe, brain lesions were visible to the naked eye. The lesions varied considerably in size and extent, but were mainly yellowish, slightly raised areas of necrosis in the cerebral cortex. In 2 lambs similar areas were also present in the cerebellum. These necrotic areas were bilateral but not necessarily symmetrical although quite often corresponding gyri were affected. Associated oedema, although not constant, was frequently seen. Correlation of brain damage with the length of illness was difficult but in a number of acute cases, these dying within 24 hours of first showing signs, the lesions were small and in the majority of cases dying after 2 to 6 days they were extensive and more diffuse in nature. The lesions in the lambs which recovered were not extensive and consisted of small localised areas of depressed and discoloured tissue

* See Addendum.

on the dorsal or dorso-lateral aspects of the cerebrum (Fig. 5). Examination of the cut surface showed that the necrosis was confined to the cerebral cortex with a clear line of demarcation between the affected cortex and the underlying white matter and also between affected and normal cortex (Fig. 6). The macroscopical appearance of the brains of affected ewes was similar to that of the lambs.

Bacteriology

Aerobic and anaerobic cultures on 5 per cent. sheep blood agar prepared from brain, heart blood, liver, small intestine and mesenteric lymph glands did not reveal any significant organisms. Portions of affected brain tissue were incubated in glucose broth at room temperature for the detection of *Erysipelothrix monocytogenes* but this organism was never isolated. Tests for the presence of mouse lethal toxin in intestinal contents were carried out, but it was demonstrated only on 3 occasions. By the Gel diffusion precipitin technique, however, of 48 samples examined 32 gave a reaction against *Clostridium welchii* Type D antiserum.

Other examinations

Biochemical analysis of blood and serum samples for calcium, magnesium, phosphorus, copper and lead gave consistently normal results. Routine examinations of samples of cerebrospinal fluid from 12 typical cases has not shown any constant abnormality. Haematological examinations were carried out on 8 cases. An increase in the packed red cell volume was noted in all, including 2 lambs which eventually recovered. In 5 others there was in addition a definite neutrophilia.

Transmission experiments

Emulsified brain tissue was injected intraperitoneally into mice, guinea-pigs and lambs; serum and cerebrospinal fluid from affected lambs was also injected intravenously into mice, all without apparent effect. Following the suggestion of Hartley in 1956 that *Cl. welchii* Type D toxin may have been associated with focal symmetrical encephalomalacia in lambs, doubling daily doses of a trypsinised *Cl. welchii* Type D toxin prepared from a field strain, were injected intravenously into 2 lambs. One lamb died during the night following the third dose of toxin equivalent to 4 mouse LD50's and the *post-mortem* examination carried out the following day failed to reveal any lesions similar to cerebrocortical necrosis. The second lamb was killed in apparently normal health 10 days later, after it had received a total of 8,194 mouse lethal doses. No discernible lesions were present on *post-mortem* examination and the brain appeared normal when examined histologically. (See Table I.)

The Disease in Calves

Twelve outbreaks of a similar condition in calves 3 to 7 months old were investigated between February, 1958, and March, 1960.

History

The 11 farms involved were spread throughout East Anglia in 5 counties. Altogether 39 calves showed signs of the disease and detailed laboratory examinations were carried out on 24. Five breeds and their crosses were involved and heifer calves, bull calves and castrated calves appeared equally susceptible. All the calves were between 3 and 7 months old, the majority being about 5 months.

The incidence varied from 1 to nearly 50 per cent., the highest proportional loss being in a small group of 13 calves where 6 were affected. On 2 farms, each with approximately 150 calves, 9 and 6 calves were affected. Although losses usually occurred within a period of 3 to 4 weeks, only rarely was more than one calf affected at the same time. Outbreaks occurred at all times of the year, though 8 of those investigated were encountered during the winter months November to March.

On only 1 of the 11 farms have outbreaks occurred in successive years, where 6 calves were affected in June, 1959, and 3 in June, 1960.

Management

With 2 exceptions, the calves were housed; the buildings varied from good loose-boxes to improvised straw bale barns. In some outbreaks several cases occurred in a single box or yard while in others single animals were affected in different enclosures on the same farm. The 2 outbreaks affecting calves which were not housed were on the same farm. In 1959 the calves were grazing a new ley and in 1960 were grazing a permanent pasture; they received oats and proprietary calf pencils in addition.

Generally the feeding methods employed were conventional for the part of the country and consisted of hay and a mixed concentrate ration, mostly oats, flaked maize and barley. In addition, proprietary calf cubes were also fed on 4 of the farms. Home-grown potatoes were fed on one farm and pea silage on another. On 4 of the farms the calves were home-bred and received some whole milk up to 6 to 8 weeks of age. The remainder were purchased calves and were reared on early weaning systems using proprietary milk substitutes. With the exception of one farm water was readily available from springs, wells or mains supply. On the one farm where water was restricted sewage contamination had apparently made it unpalatable. Bedding varied, but was usually wheat or oat straw. Investigation into spraying and fertiliser programmes of all home-grown crops did not reveal any consistent features.

Clinical appearance

All the calves showed nervous signs and characteristically these became progressively more severe as time elapsed. At first the calves were dull and did not feed. Apparent blindness, staggering, tetanic spasms and fits often followed within a few hours. Usually 12 to 72 hours after the onset of signs they became recumbent. If propped up their heads appeared too heavy to be supported and often developed a weaving motion; their ears drooped.

TABLE I
SUMMARY OF OUTBREAKS IN SHEEP

Outbreak	Date	Age	Breed	Incidence	Supplementary Food	Pasture	Enterotoxaemia vaccination
1	April 1959	8 weeks	Southdown	3/150	Oats and beet pulp with mineral supplement	Folded on rape	At 4 weeks
2	April-June 1959	6–12 weeks	Suffolk × half-bred	23/470	Crushed oats and mineral supplement	Folded on second year leys (lush)	At approx. 4 weeks
3	May 1959	7 weeks	Suffolk × half-bred	1/288	Crushed oats, barley, beet pulp, linseed cake and mineral supplement	Permanent pasture	None
4	May-June 1959	8–12 weeks	Suffolk × half-bred	17/450	Crushed oats and mineral supplement	Folded on new leys	At approx. 4 weeks
5	May–June 1959	10–16 weeks	Suffolk × half-bred	3/300	Mangolds, oats, beet pulp and mineral supplement	Folded on new leys	Lamb dysentery antiserum at birth
6	June–July 1959	12–16 weeks	Suffolk × Border Leicester	17/250	Oats and beet pulp with mineral supplement	Semi-permanent pasture and new leys	At 4–6 weeks
7	June–July 1959	12–16 weeks	Suffolk × half-bred	2/470	Proprietary lamb cubes	Folded on new leys	At approx. 4 weeks
8	April–May 1959	6–8 weeks	Suffolk × half-bred	8/487	Oats, rock salt and mineral licks	Folded on leys	Lamb dysentery antiserum at birth
9	April–May 1959	8–12 weeks	Suffolk × half-bred	15/500	Oats and mangolds	Folded on leys	None
10	May 1959	12 weeks	Suffolk × half-bred	5/400	Proprietary pellets, pulp and oats with mineral supplement	Folded on leys	None
11	May 1959	12 weeks 2 years	Suffolk × half-bred lambs Half-bred ewes	8/120 lambs 3/60 ewes	Oats, beet pulp and proprietary nuts	Permanent pasture and leys	None
12	July 1959	2 years	Half-bred ewes	1/80	Proprietary cow food	Permanent grass	Ewes before lambing
13	September 1959	6–7 months	Suffolk × half-bred	2/400	None	Folded on leys	None
14	May 1960	7–12 weeks	Suffolk × half-bred	12/480 2 recovered	Oats, mineral licks	Folded on leys, trefoil and red clover	At 4 weeks
15	May 1960	6–8 weeks	Suffolk	7/50	Oats, mineral licks	Folded on 4-year ley	None
16	May 1960	6–8 weeks	Suffolk × border Leicester	1/500	Oats, barley, sugar beet pulp, mineral licks	Semi-permanent and permanent pasture	At 7 weeks
17	June 1960	2–7 years	Clun Forest ewes	6/120 ewes	Barley, wheat, beans, linseed cake and mineral supplement	2-year lucerne and cocksfoot leys	Ewes before lambing
18	June 1960	8–12 weeks 2–7 years	Suffolk × Border Leicester lambs Border Leicester ewes	7/250 lambs 8/165 ewes	Oats, sugar beet pulp and mineral supplement	2-4 year leys and permanent grass	Ewes before lambing, lambs at 5 weeks
19	June 1960	12 weeks	Suffolk × half-bred	1/470	Proprietary lamb and ewe food	First year ley, ryegrass, fescue, clover	Ewes before lambing, lambs at 5 weeks
20	June 1960	12 weeks	Suffolk × half-bred	1/400	Proprietary food, home grown oats, beans and mineral supplement	4 and 2 year ley of ryegrass, Timothy and clover	Ewes before lambing, lambs at 4 weeks
21	June 1960	12–16 weeks	Suffolk × Border Leicester	6/400	Oats, locust bean and mineral supplement	Folded on six-year ley	Ewes before lambing, lambs at 5 and 10 weeks

In the final stages the limbs and head were extended (Fig. 7) but in contrast to tetanus the limbs could be flexed manually. Other features frequently present included a brown diarrhoea, general muscular twitching or twitching of the ears and eyelids and grinding of the teeth accompanied by groaning. A sudden noise or other stimulus sometimes produced excitement, but this was not as common as in sheep. Death usually occurred within 3 days of the onset of signs, but some calves remained alive up to 6

days and were killed *in extremis*. Throughout the illness the temperatures usually remained normal.

To our knowledge only 5 calves recovered after showing typical signs; 2 of these were kept under observation at the Cambridge Veterinary Investigation Centre and showed gradual improvement. One of these (A) was killed after 2 months when it was impossible to detect any clinical abnormality. The other (B) was killed 2 weeks after first showing nervous signs when it was still unsteady and only mouthing its food and water. The remaining 3 came from the same farm as (B) and in them signs of the disease disappeared 2 to 3 weeks after the onset of illness (Fig. 8). After an initial setback these animals appeared to thrive.

Treatment

On most farms various antibiotics and calcium and magnesium injections were commonly tried but without success. Treatment with chloramphenicol, on the farm where 3 calves recovered, appeared to be effective. However, there was no response in affected calves treated similarly on other premises.

Post-mortem *findings*

The specimens examined were 13 calves received at the laboratory alive and showing severe clinical signs, 2 calves freshly dead and the heads and a variety of other viscera from 9 typical clinical cases. One of the live calves died 48 hours after arrival at the laboratory, the remainder were killed by injection of magnesium sulphate or pentobarbitone sodium intravenously.

Lesions in organs other than brain were seen in only 3 calves, namely the one which died after arrival at the laboratory and the 2 which were brought in dead. The first of these was affected with chronic pneumonia, the second had some ulceration of the abomasum and the third had slight cirrhosis of the liver.

In all cases it was possible to detect some gross brain lesions. Characteristically, these consisted of yellow-brown necrotic areas in both cerebral hemispheres. The same region of each hemisphere was usually affected, but the extent varied. In the majority there was a distinct line of demarcation between diseased and normal tissue, but in a few the lesions were more extensive and diffuse. The affected areas were usually swollen and soft to the touch with considerable oedema in the sulci. In the brain of the partially recovered calf (B) the lesions were unusual in that they were depressed and showed evidence of cavitation. In 4 cases there was also a mild meningitis. No macroscopical lesions were seen in the spinal cords of any of these cases.

Bacteriology

Routine bacteriological examination of brain and other organs was carried out using 5 per cent. sheep blood agar and incubating aerobically and anaerobically. Cultures prepared from 15 brains immediately after removal from calves killed for *post-mortem* examination remained sterile after prolonged incubation. A variety of bacteria, predominantly streptococci, staphylococci and *E. coli*, were recovered from 7 specimens received in less fresh condition. In the outbreak where meningitis occurred *C. pyogenes* was recovered from the meninges of 2 calves.

Biochemistry

In attempts to differentiate the condition from lead poisoning at least one kidney from each outbreak was examined chemically. Altogether 16 kidneys were examined without detecting any abnormal lead content. In addition, blood and faeces samples were collected from in-contact calves on two farms, but again analysis failed to reveal the presence of lead at abnormally high levels.

Blood samples taken from typical cases in each

TABLE II
SUMMARY OF OUTBREAKS IN CALVES

Outbreak	Date	Area	Age	Breed	Incidence	Type of management
1	February-April 1958	Norfolk	4-5 months	Friesian and Hereford ×	9/150	Purchased calves in pens and yards
2	June 1959	Essex	4-6 months	Ayrshire	6/13	Home-bred calves grazing ley
3	June 1959	Suffolk	6 months	Cross-bred	2/5	Purchased calves in straw yards
4	July 1959	Cambridgeshire	4 months	Friesian	2/7	Purchased calves in barn
5	October-November 1959	Norfolk	5 months	Friesian × Shorthorn	2/18	Home-bred and purchased calves in loose-boxes
6	January 1960	Essex	3 months	Hereford	1/80	Purchased calves in straw pen in barn
7	December-February 1959-60	Norfolk	6 months	Friesian	3/14	Home-bred calves in barn
8	January 1960	Cambridgeshire	4 months	Friesian	2/30	Home-bred calves in yards and open boxes
9	January 1960	Norfolk	3-4 months	Friesian	4/28	Home-bred calves in yards
10	February 1960	Cambridgeshire	5 months	Aberdeen Angus ×	2/20	Purchased calves in loose-boxes
11	February-March 1960	Northampton-shire	5-6 months	Friesian × Hereford	6/150	Purchased calves in yards
12	June 1960	Essex	4-7 months	Ayrshire	3/26	Home-bred calves grazing old pasture

S. TERLECKI & L. M. MARKSON.—CEREBROCORTICAL NECROSIS IN CATTLE AND SHEEP

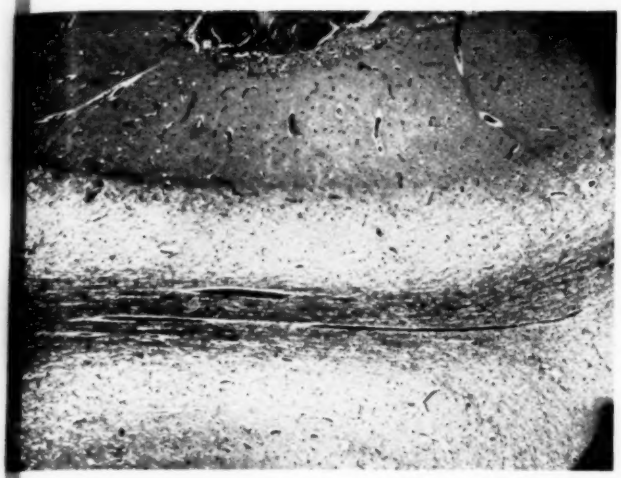

FIG. 1.—Cerebrum of an ox. Laminar microcavitation on each side of gyral white matter Heidenhain's iron haematoxylin. x 27 approximately.

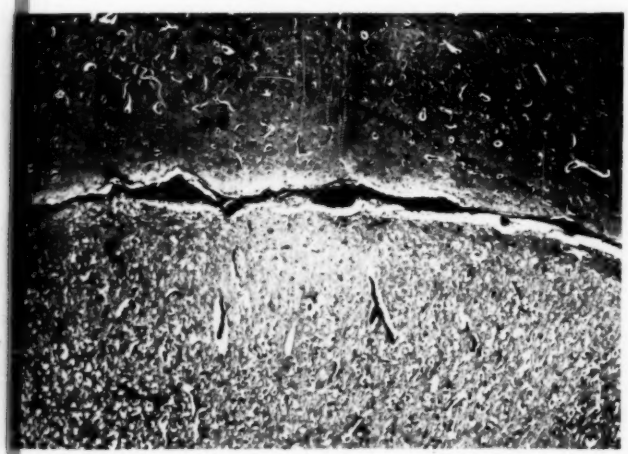

FIG. 2.—Cerebrum of an ox. The lower gyrus shows microcavitation of the superficial cortex. Heidenhain's iron haematoxylin. x 36.

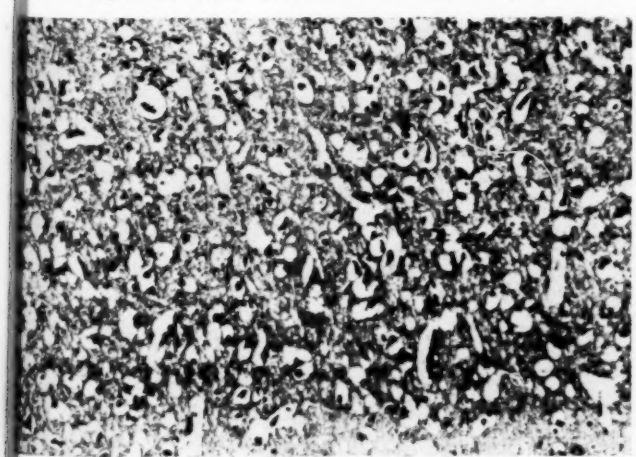

FIG. 3.—The same section as FIG. 1 photographed at a higher magnification to show shrunken neurons and perineuronal vacuolation. x 45.

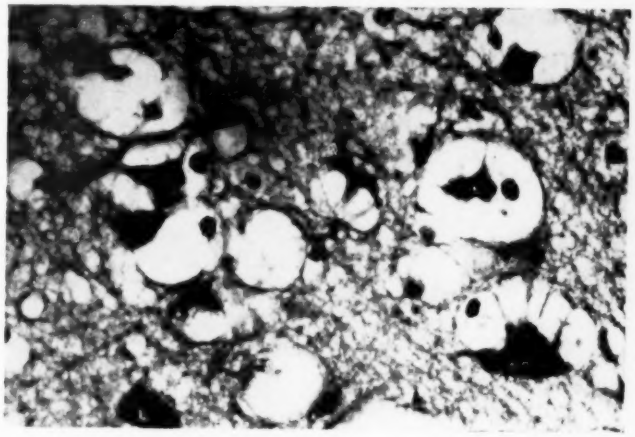

FIG. 4.—A contiguous section from the same block illustrating shrunken and necrotic neurons. Haematoxylin and eosin. x 450.

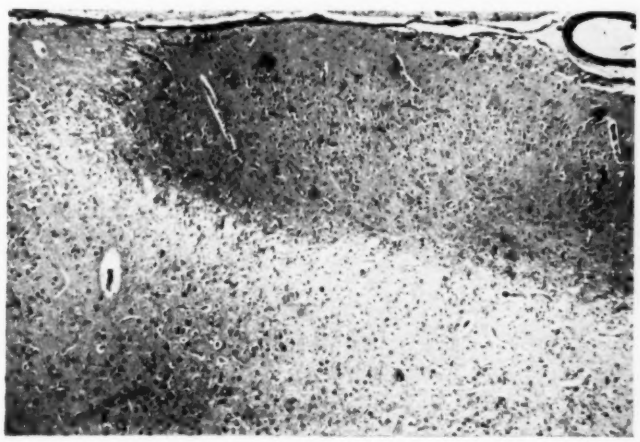

FIG. 5.—Cerebrum of a sheep. An area of superficial cortex with necrosed neurons set in a hyperchromatic matrix. Microcavitation is apparent in the surrounding tissue. Haematoxylin and eosin. x 45.

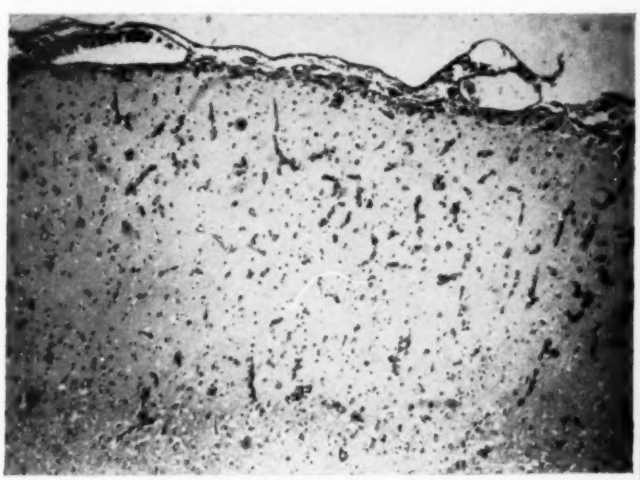

FIG. 6.—Cerebrum of an ox. Slight meningeal reaction and prominent capillary vasculitis in the superficial cortex. Haematoxylin and eosin. x 45.

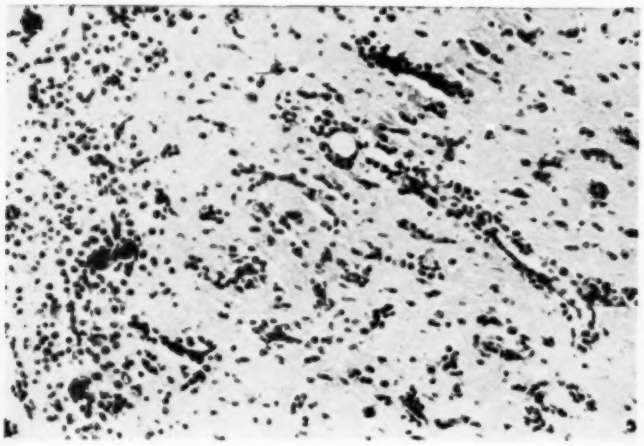

FIG. 7.—Cerebrum of an ox. Severe meningeal infiltration by macrophages with invasion of the cortex by extension from the surface and by perivascular spaces. Haematoxylin and eosin. x 135.

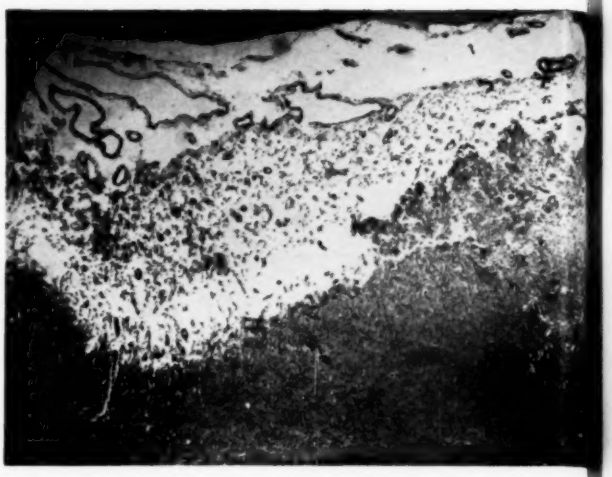

FIG. 10. Cerebrum of an ox two weeks after the onset of symp Local disintegration of the necrosed cortex with formation of a meningeal cavity. Heidenhain's iron haematoxylin. x 27.

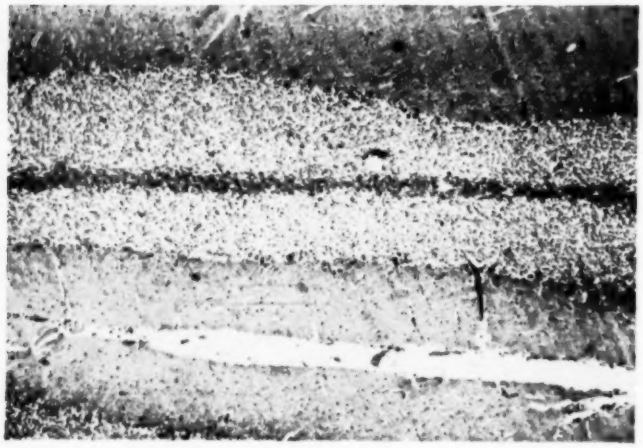

FIG. 8. Cerebellum of an ox. Severe rarefaction of the granule cell layer. Haematoxylin and eosin. x 45.

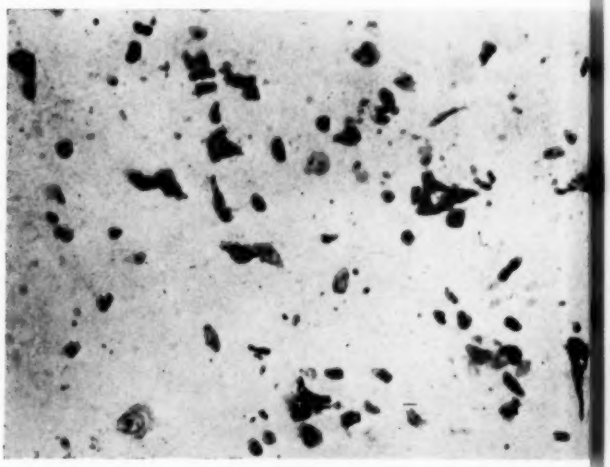

FIG. 11.—Cerebrum of a sheep. Calcified neurons. Von Kossa's met x 450.

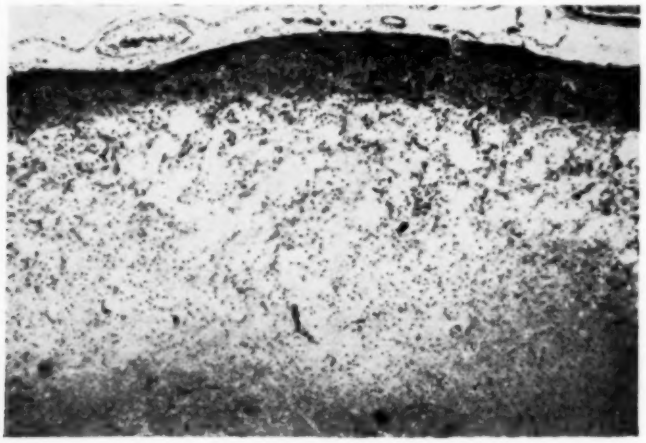

FIG. 9.—Cerebrum of a sheep 10 days after the onset of symptoms, showing an advanced lesion with disintegration of the necrosed cortex. Heidenhain's iron haematoxylin. x 45.

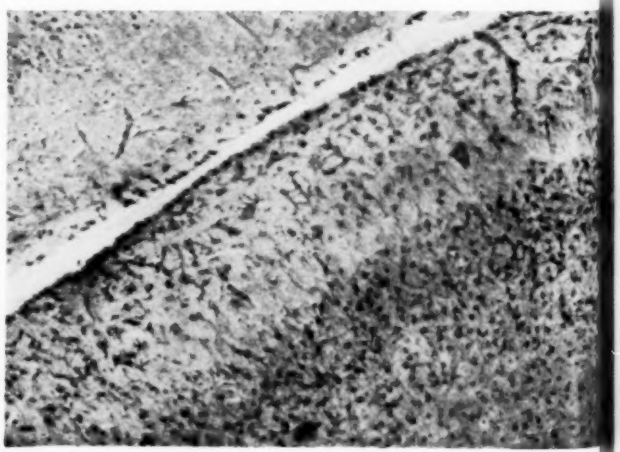

FIG. 12.—Cerebrum of a recovered sheep. Laminar astrocytosis. small segment of the contiguous gyrus is apparently normal. Ca gold chloride-sublimate method. x 45.

FIG. 1.—A 2-month-old Suffolk x Half-bred lamb affected for 2 days.

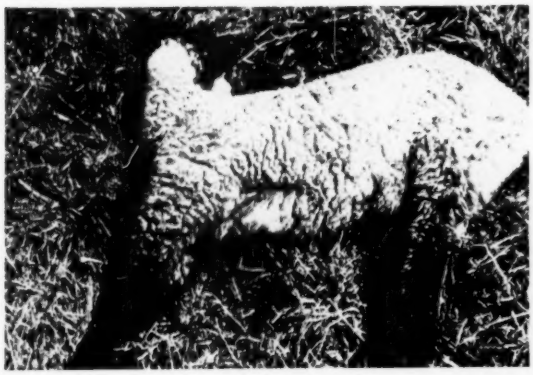

FIG. 2.—A 2-month-old Suffolk x Half-bred lamb showing a tetanic spasm.

FIG. 3.—A 7-year-old Border Leicester ewe affected for 24 hours.

FIG. 4.—A 4-year-old Clun Forest ewe affected for approximately 12 hours.

FIG. 5.—Brain from clinically recovered 3-month-old Suffolk x Half-bred lamb killed 10 weeks after first showing signs.

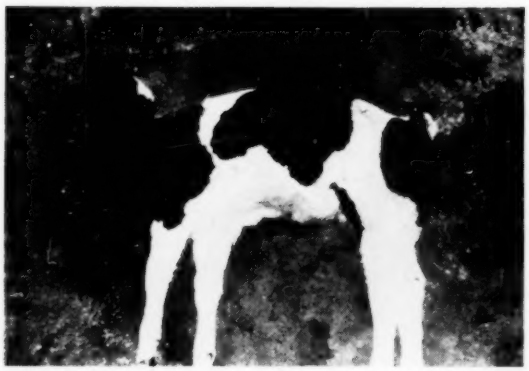

FIG. 7.—A 4-month-old Friesian calf showing opisthotonos.

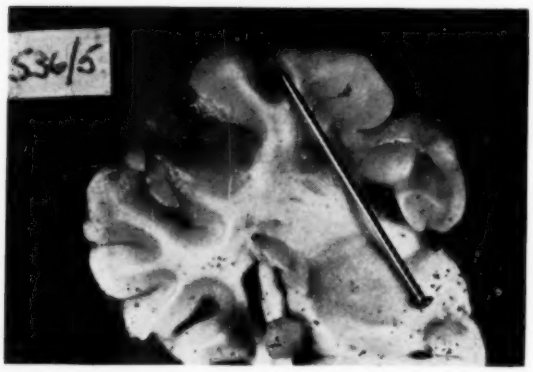

FIG. 6.—Cross section of cerebrum from 2-month-old Suffolk x Half-bred lamb showing cortical necrosis and line of demarcation between affected and normal tissue.

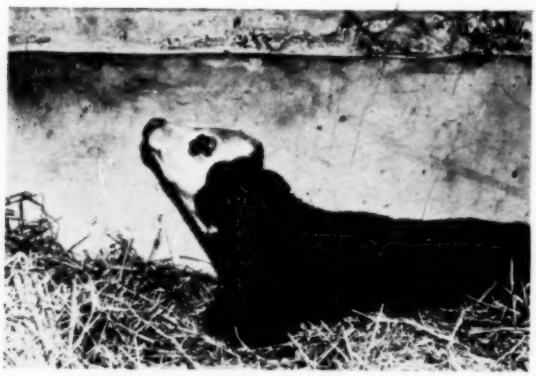

FIG. 8.—A 4-month-old Hereford calf affected 24 hours, this case recovered clinically in 3 weeks.

outbreak and examined for calcium, magnesium, phosphorus, acetone and copper, did not show any consistent abnormality. Livers from 3 typical cases were examined for their vitamin A content; the results ranged from 15 to 32 international units per gramme and were considered normal.

Transmission experiments

The following small scale transmission trials were carried out. Emulsified brain tissue from 3 typical cases failed to infect mice when injected intraperitoneally. Samples of hay and various concentrate rations were collected from 4 farms and fed to guinea-pigs for periods varying from 10 days to 4 weeks without effect. On one farm it was suspected that home-grown barley incorporated in the calf ration had been dressed with dieldrin. A sample of this barley was fed to 3 pigeons for 3 weeks without producing any visible abnormalities.

Discussion

During the past 6 years a number of reports have appeared in the literature of diseases in sheep and cattle affecting the central nervous system in which the predominant lesion was necrosis. In none of these cases has the exact cause been determined.

In 1954 Plowright described a focal and symmetrical spinal poliomalacia in sheep in Kenya, the result, he suggested, of an unknown toxic factor leading to a conditioned deficiency of a metabolite essential for the normal functioning of the spinal grey matter. Focal symmetrical encephalomalacia of lambs in New Zealand was described by Hartley in 1956 who, when discussing its possible cause, suggested that a sublethal dose of *Cl. welchii* Type D toxin may have been involved. This theory received some support from the work of Gill (1927-33). Jensen, Griner and Adams (1956) described a polioencephalomalacia of cattle and sheep in the U.S.A. which they attributed to an unknown intoxication. Mullins, Hartley and Salisbury (1958) recorded an outbreak of blindness in sheep in New Zealand where lesions were confined to the cerebral cortex; many sheep were affected, but the mortality rate was low. Hartley and Kater (1959) reported a polioencephalomalacia in sheep in New Zealand in which unspecified toxins were thought to be involved. They considered the blindness syndrome described by Mullins *et al.* (1958), to be a mild form of polioencephalomalacia. Barlow (1958) and Robertson and Wilson (1958) reported outbreaks of focal symmetrical encephalomalacia in sheep in Great Britain.

Sporadic cases of cerebrocortical necrosis in sheep have been recognised in Great Britain since 1954 by Terlecki and Markson (1959). The outbreaks described in this paper are, however, the first recorded in Great Britain where the incidence and mortality have reached serious proportions. It seems highly unlikely that the syndrome would have escaped notice in the past if similar epidemics had occurred.

The possible causes put forward by the workers mentioned above have been considered and it is agreed that the evidence would most strongly suggest that some form of toxic factor is involved. The epidemiological picture and the bacteriological examinations of the outbreaks in East Anglia do not support the hypothesis that *Cl. welchii* toxins are connected with cerebrocortical necrosis. A viral aetiology seems unlikely in view of the non-inflammatory nature of the brain lesions and the failure to transmit the disease experimentally. At present, however, the possibility of either a bacterial toxin or a virus being the primary cause of the condition cannot be entirely ruled out. Poisonous plants, or indeed common weeds, were largely absent from the pastures grazed by the sheep and all but one of the cattle outbreaks occurred in housed calves where the food supplied was apparently of good, normal quality. The first series of outbreaks in sheep was encountered during the particularly hot, dry spring and summer of 1959 and the possibility of water deprivation was considered especially because of the comparable lesions described by Done, Harding and Lloyd (1959) in experimental salt poisoning of pigs. In only one of the calf and in one of the sheep outbreaks was there thought to be a shortage of water. The association of fungal toxins with maize stalk poisoning in horses, Rodriguez (1945), facial eczema in sheep, Thornton and Percival, (1959), a convulsive syndrome in sheep, Clegg (1958) and Temblique in cattle (unpublished data, Commonwealth Bureau of Animal Health) suggested another line of investigation, but so far no evidence has been obtained to support this theory. Finally, careful consideration was given to chemical poisons, particularly those which during the last few years have become so extensively used in modern agriculture. Though on nearly all the farms weed killer sprays had been used for some time there was no particular type of spray common to all the outbreaks and in very few cases had there been recent applications which might have led to direct contact with quantities of chemical. In the calves access would have had to have occurred through hay or bedding and in no cases did this possibility seem likely. There was no evidence that the concentrate foods contained dressed seed and small feeding trials using rodents and pigeons failed to reveal any of the usual substances.

Whilst a toxic factor seems the most likely cause of cerebrocortical necrosis it is difficult to explain the unusual seasonal incidence in sheep, the simultaneous occurrence of similar outbreaks in calves, the predilection for young animals of a specific age group and the exceptional epidemics of the last two years. A study of the epidemiology seems to be the most promising line of investigation, but so far it has not been possible to define a specific cause.

Acknowledgments.—We wish to thank S. Terlecki and L. M. Markson for drawing our attention to their previous observations and for examining and reporting on the tissues sent for histological examination. We also wish to thank the veterinary surgeons in private practice and farmers who have supplied information and material. Finally, we wish to acknowledge the help received from many

(*Concluded at foot of col. 1 overleaf*)

The Acceptability of Calcined Magnesite to Cattle with a Note on the Response Obtained in a Case of Hypomagnesaemia

BY

RUTH ALLCROFT and ANNIE I. LITTLEJOHN

Central Veterinary Laboratory, Weybridge, Surrey

THE value of oral magnesium supplements for the prevention of hypomagnesaemia was demonstrated in 1935 by Blakemore and Stewart who fed a magnesium-rich mineral supplement mixed with a small amount of cake in troughs to cows grazing spring "tetany-producing" pastures. The mineral supplement contained 10 per cent. magnesium in the form of magnesium oxide as well as other minerals. In 1938, Allcroft and Green reported that access to a mineral mixture containing about 50 per cent. magnesium oxide present as calcined magnesite, when offered in troughs, partially alleviated the hypomagnesaemia that occurred in grazing Hereford cows from October to December.

During the 15 years following these publications, conflicting reports were received from veterinary surgeons regarding the value of oral supplements of magnesium-rich mineral mixtures, both for the prevention of hypomagnesaemic tetany and as supportive treatment following the usual parenteral administration of magnesium sulphate to clinical cases. In the many instances where mineral mixtures were reported to be ineffective in preventing hypomagnesaemia, no information was given regarding the magnesium content of the mixtures or the amount consumed by the animals. Later work (Allcroft, 1954) demonstrated that a daily dose of 2 oz. magnesium oxide (B.P. heavy grade) given as a drench to milking cows grazing a "tetany-producing" pasture, or fed in troughs mixed with a little crushed oats to outwintered dry stock, would prevent hypomagnesaemia. Subsequent observations at the National Institute for Research in Dairying and at Weybridge have shown that 2 oz. magnesium oxide daily is approximately the minimum effective prophylactic dose for adult cattle.

Because of the high cost of a B.P. grade of magnesium oxide, a commercial grade ("calcined magnesite"*) containing 87 to 90 per cent. magnesium oxide has been generally used in magnesium-rich commercial mineral mixtures. It has been found at Weybridge (unpublished data) that 2 oz. of this grade was as effective as 2 oz. of B.P. heavy grade magnesium oxide.

In many herds it is not the practice to give either concentrates or other mineral supplements to certain classes of stock which nevertheless require prophylactic treatment against hypomagnesaemic tetany. Moreover, during the last few years, it has frequently been claimed that one of the chief objections to the use of calcined magnesite either alone, or mixed with or sprinkled on feeding-stuffs such as cereals, chopped roots or silage, is its unpalatability. We have not found this to be so during its frequent use over the last 7 years in 2 experimental dairy herds kept under close observation. It is considered that initial refusal by a few animals may have been attributable to bad mixing with the foodstuffs and to the powdery nature of finely ground calcined magnesite, but no evidence of unpalatability (as opposed to unacceptability) has been observed.

Recently we had the opportunity to investigate the question of acceptability in a group of 44 heifers under more uniform conditions of management than

*Large deposits of magnesite, *i.e.* magnesium carbonate, occur in certain parts of the world. Heating, or calcining, produces calcined magnesite, which usually contains 87 to 90 per cent. magnesium oxide, and is imported into this country for industrial purposes. A finely ground, powdery form of this commercial grade magnesium oxide is used extensively in mineral mixtures or incorporated into pelleted foods for stock.

Cerebrocortical Necrosis in Sheep and Cattle: The Clinical Syndrome.—*Concluded.*

colleagues in the Ministry of Agriculture Veterinary services and the University of Cambridge and for the technical assistance given by the Biochemical Department, Weybridge, and the Departments of Clinical Pathology and of Photography, Cambridge.

Addendum

Since submitting this paper for publication histological examination of the brain of one of these ewes has shown evidence of listeriosis only.

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are found in a commercial milking herd. As a granular form of calcined magnesite was also available the acceptability of this was compared with that of the more commonly used powder form. The results of the observations are given below.

Materials and Methods

The animals used were 20- to 27-month-old Ayrshire heifers, between 90 and 120 days pregnant, accustomed to being stall-fed and handled. They comprised (Trials 1 to 5) the 15 largest and the 15 smallest of a group of 60 heifers recently discharged from a nutritional experiment in which they had been receiving for the preceding 8 months, and were still receiving, a ration consisting of damp sugar-beet pulp, ground-nut cake and oat straw, and (Trial 6) 14 heifers of comparable age and stage of pregnancy which had been fed during the same period on the ordinary Weybridge cattle diet of hay, greenstuff and compound nuts.

As all the heifers in each group received the same quantity of food irrespective of bodyweight, the largest heifers were in the habit of eating their rations more eagerly than were the smallest animals.

All but 6 of the heifers in the first group were accustomed to having a calcium and/or phosphorus supplement mixed into their ration of damp sugar-pulp every day. No mineral additions had at any time been made to the rations of the heifers in the second group.

Although the normal rate and manner of feeding of each heifer were well known to one of us (A.L.) who had had the animals continually under observation during the preceding 8 months, each of the 2 preparations, *i.e.* powdered and granular calcined magnesite, was in any one trial given on the first day to only half of the heifers in the group concerned, the other half of the group receiving the other preparation in order to permit a simultaneous comparison of the rates of feeding. On the following day the halves were reversed.

Two ounces of calcined magnesite, either as a finely ground powder or as $\frac{1}{8}$ to $\frac{1}{4}$ inch granules were added to three different types of feeding-stuffs in the following way:—

1. Well mixed* with damp sugar-beet pulp (7½ lb.)
2. Badly " " " " " " "
3. Well " " dry† " " " "
4. Badly " " " " " " "
5. Well " " ground-nut cake (1½ lb.) "
6. " " " compound nuts (3½ lb.) "

*Mixing was carried out after the ration had been placed in the manger.

†This trial was carried out after the heifers had become accustomed to receiving their ration of sugar-beet pulp dry instead of damp.

Results

Trial 1 (Damp sugar-beet pulp, well mixed, 30 heifers)

Neither the powdered nor the granular magnesite affected the rate of feeding of any of the larger heifers. Some of the smaller heifers were a little slower than usual in clearing up their ration but this was not related to the form in which the magnesite had been given.

Trial 2 (Damp sugar-beet pulp, badly mixed, 30 heifers)

In this trial the magnesite was placed on top of the damp sugar-beet pulp and spread roughly over the top of the ration with 2 movements of the hand. All but the greediest feeders were a little slower than usual in finishing the ration, and this was more noticeable when the powdered form was fed. In no instance was any of the sugar-beet pulp left in the manger, but when powder was fed, several of the small heifers and one of the large heifers, contrary to the usual practice of all the heifers, failed to lick the manger clean, leaving untouched those places on to which a pile of powder had fallen while the sugar-beet pulp was being eaten. When the granular form was fed, however, the manger was licked clean in the usual way although, as already mentioned, some of the heifers took longer than usual to finish the ration.

Trial 3 (Dry sugar-beet pulp, well mixed, 30 heifers)

By the time that this trial took place, the animals had become accustomed to receiving their sugar-beet pulp ration dry and all but 3 of the smaller heifers were in the habit of clearing it up at once. In this trial there was a noticeable difference between the acceptability of the 2 forms of magnesite. When the granules were fed, the manger was licked clean as usual, except by 2 of the smallest heifers which left a few granules, whereas when the powder was fed, almost all the heifers left a small pile of sugar-beet pulp dust, containing varying amounts of the powdered magnesite.

Trial 4 (Dry sugar-beet pulp, badly mixed, 30 heifers)

Results in this trial emphasised the difference in acceptability between the granular and powdered forms. Only 2 of the smaller heifers receiving the granular form failed to lick the manger clean whereas large patches of powder or small piles of powder and sugar-beet pulp dust were left by all but one of those receiving the powdered magnesite.

It may be mentioned here that in the group of 30 heifers receiving the magnesite in the sugar-beet pulp there was no difference in appetite between the 24 heifers accustomed to receiving a Ca and/or P supplement well mixed with the sugar-pulp beet and the 6 heifers that had not previously received a supplement.

Trial 5 (Ground-nut cake, well mixed, 30 heifers)

When the granular form was fed all but one of the heifers, which licked the manger clean, left amounts varying from a few granules to approximately 20 per cent. of the amount of magnesite fed. Two also left a small amount of cake uneaten. Of those receiving the powdered form, one licked the manger clean, but all the others left large patches of powder in the manger, and 4 of these also left some of the ground-nut cake uneaten. It was not possible to measure the amount of powder left, but the general impression was that it represented a considerably greater proportion of the amount fed than did the granules remaining in the mangers.

Trial 6 (Compound nuts, well mixed, 14 heifers)

The heifers used in these trials were unaccustomed to receiving mineral supplements of any kind, and all but one of them took a little longer than usual to finish the ration. When the granules were fed, the mangers were without exception licked clean in the usual manner, whereas when the powder was used, all the heifers, except one greedy feeder which licked the manger clean, left amounts of powder varying from 20 to 90 per cent. of the original quantity fed.

Conclusions

When calcined magnesite is thoroughly mixed with damp sugar-beet pulp, it is readily eaten whether in powder or granular form. When mixing is inadequate or when the foodstuff used as a vehicle is dry, the granular form is much more acceptable, the amount rejected not exceeding 20 per cent. of the granular form but reaching 90 per cent. of the powder. The difference between the 2 groups in Trial 6 (with compound nuts) was particularly striking.

Response to the Oral Administration of Granular Calcined Magnesite in a Case of Hypomagnesaemia

During the course of the nutritional experiment referred to above, a case of subclinical hypomagnesaemia was noted in one of the experimental heifers. This animal was receiving a daily ration of 7¼ lb. sugar-beet pulp, 1¼ lb. ground-nut cake and 10 lb. of oat straw, plus a mineral supplement which raised the total daily intake per 100 lb. bodyweight to 15 g. CaO and 12 g. P_2O_5. There had been no disturbance in general health; in fact the heifer, although small for her age (20 months), was thriving particularly well, having increased in weight from 421 lb. in the middle of April to 613 lb. in the middle of October, when treatment began. She had held to the first service on August 25th.

In view of the steady fall observed in serum magnesium it was decided on October 13th to begin the administration of a magnesium supplement.

Two ounces of granular calcined magnesite were thoroughly mixed with the damp sugar-beet pulp in which the supplementary calcium and phosphorus were also being fed. The heifer continued to eat the now heavily supplemented ration with good appetite. Blood samples were taken at frequent intervals for estimation of serum magnesium and calcium; the tabulated results are given below.

As may be seen, the response to the oral administration of daily doses of granular calcined magnesite was very satisfactory. In 33 hours the serum magnesium level had risen from 0.9 to 1.6 mg. per 100 ml. and a normal level (1.8 mg. per 100 ml.) was reached 57 hours after the start of the treatment. The level was still satisfactory (1.8 mg. per 100 ml) 33 days after the magnesite had been withdrawn from the diet.

It may be noted that although the serum calcium remained within the accepted normal range, there was a tendency for the lower levels to occur during the period of most pronounced hypomagnesaemia and for the higher levels to coincide with the return of the magnesium levels to normal.

EFFECT ON SERUM MAGNESIUM AND CALCIUM LEVELS OF FEEDING 2 OZ. OF GRANULAR CALCINED MAGNESITE DAILY FOR 10 DAYS

Date of blood sample	2 oz. granular calcined magnesite fed	Magnesium mg. per 100 ml.	Calcium mg. per 100 ml.
28th April a.m.		1·8	9·3
26th May ,,		1·8	9·8
30th June ,,		1·1	9·6
4th Aug. ,,		1·0	9·0
1st Sept. ,,		1·0	9·8
29th Sept. ,,		0·9	9·6
12th Oct. 5 p.m.		0·9	8·5
	13th Oct. 8 a.m.		
13th Oct. 5 p.m.		1·0	9·2
14th Oct. 7.45 a.m.		1·0	8·7
	14th Oct. 8 a.m.		
14th Oct. 5 p.m.		1·6	10·4
	15th Oct. 8 ,,		
15th Oct. 5 ,,		1·8	10·1
	16th Oct. 8 ,,		
16th Oct. 5 ,,		2·0	10·0
	17th Oct. 8 ,,		
	18th Oct. 8 ,,		
18th Oct. 5 ,,		2·0	9·7
	19th Oct. 8 ,,		
	20th Oct. 8 ,,		
20th Oct. 5 ,,		2·0	10·1
	21st Oct. 8 ,,		
	22nd Oct. 8 ,,		
22nd Oct. 5 ,,		2·1	10·1
27th Oct. a.m.		1·7	8·9
24th Nov. ,,		1·8	11·0

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RUSSIAN ENTRANTS TO BRITISH HORSE RACING

In view of forecasts in some newspapers that horses from the U.S.S.R. may be entered in British events during the coming year, the following note, circulated by the Ministry of Agriculture under the title African Horse Sickness, is timely.

The responsibility for preventing the introduction of animal diseases into this country rests with the Secretary of State for Scotland and the Minister of Agriculture. They have the benefit of information collected by international organisations.

African Horse Sickness is endemic in parts of Africa, and since October, 1959, has spread to a number of near and middle eastern countries. It is carried by insect vectors and is a deadly disease. Morbidity and mortality rates often approach 100 per cent.

Information as to the countries from which imports of horses carry danger of horse sickness was supplied to the Government by the International Office of Epizootics. This list included the U.S.S.R. So long as any country is included in the Order the ban on horses from it is absolute. The list of countries is reviewed at intervals and if we are satisfied that a country is out of the danger area it would be taken out of the Order.

Current Literature

ABSTRACTS

Surgery of Subcutaneous Tumours in Parakeets. GANDALL, C. P., & SAUNDERS, L. Z. (1959). *J. Amer. vet. med. Ass.* **134.** 212.

The popularity of shell parakeets and budgerigars as domestic pets is increasing in America as well as in this country. Unfortunately the development of various types of tumours is fairly common, but the authors advocate the attempted removal of any subcutaneous tumour presented for treatment. The majority of the subcutaneous type are capable of being removed successfully with a patient and proper technique. The majority of these tumours are benign in nature, but although they do not endanger the bird's life, it frequently becomes languid, unable to fly properly, and ceases talking. Of 45 tumours removed by the authors and studied histologically 5 were fibrosarcomas, 18 were lipomas, 14 were necrotic lipomas, and 8 were fat granulomas.

The authors recommend, as a general anaesthetic, the intramuscular injection of 0.22 ml. to 0.25 ml. per 100 g. bodyweight of "Equithesin" (Jensen Salsbery Laboratories). The area of the skin around the tumour is denuded by plucking the feathers and thoroughly cleaning the area with alcohol. The initial incision is made with a scalpel, but any further cutting is done with a pair of strabismus scissors. By this means there is less likelihood of accidentally incising too deeply. Each tumour is then dissected out according to its type and situation. The skin is sutured with cat gut using an atraumatic needle. The majority of birds can stand within 90 minutes following surgery, and no further treatment is required. J. S. J. L.

The Immunisation of Dairy Cattle Against Staphylococcal Mastitis. SLANETZ, L. W., BARTLEY, C. H., & ALLEN, F. E. (1959). *J. Amer. vet. med. Ass.* **134.** 155.

Cows, mainly in a herd in which staphylococcal mastitis was a serious problem, were given multiple injections of either staphylococcal toxoid or bacterin-toxoid, prepared either commercially or by the authors from a strain of *Staph. aureus* isolated from a case of bovine mastitis. An appropriate number of unvaccinated cows were kept as controls.

All methods of vaccination stimulated the production of antibodies and increased the resistance of cattle to staphylococcal mastitis. Compared with the controls, the spread of infection was almost completely prevented in the vaccinated cows and there was a marked reduction in the number of acute flare-ups in vaccinated infected cows over periods of up to 18 months.

Vaccination had little effect in eliminating infection from quarters with well-established chronic infection. H. W. S.

News and Comment

HONOUR FOR MR. THOMAS HICKS

We are indebted to Professor McCunn for the following interesting note:—

On Sunday, December 18th, and after the sermon at St. Botolphs, the Parish Church of Quarrington, Sleaford, the Rector, the Rev. H. Bailey, asked the congregation to remain in their seats for a few moments. Then Mr. Tyler, a chorister, told the congregation of a unique occasion in the history of that church. A fellow chorister, Tom Hicks, would celebrate his 81st birthday during the coming week and 50 years service in their choir. Mr. Tyler concluded by saying "Tom, we ask you to accept this portrait of yourself, in watercolour, skilfully executed by one of our members, Mr. Hedley Mobbs."

The secret had been kept so well that surprise was complete and the recipient was so moved that he could only mutter "Thank you."

Tom Hicks spent his early days as a boy chorister in Lincoln Cathedral under the tutelage of Dr. Bennett the famous choirmaster and organist. He rose to head boy and sang the solos at the choral services. When his voice broke it became that of a pure tenor, of rich quality. Since then he has sung in many churches and halls and has taken solo parts in oratorios. He has also sung principal tenor for the Sleaford Operatic Society.

It is good to know that one of our professional brothers, who has been gifted with a beautiful voice, has used it in the glory of God and the delectatio.. of his friends and neighbours. We have known Tom Hicks for many years as an extremely competent and conscientious veterinary surgeon, a good horseman, a man's man and a kind and courteous gentleman. This other side of his life may not be known to many who are privileged to call him friend, and on their behalf I say "Long may you sing in the village choir and make our hearts rejoice."

ELECTION OF R.C.V.S. COUNCIL MEMBERS

In accordance with the provisions of the Veterinary Surgeons Act, 1948, 5 members of the Council, who must be members of the Royal College, will be elected in 1961 by members of the Royal College residing outside the Republic of Ireland.

Five present members of Council will retire at the Annual General Meeting in 1961 and are eligible for re-election, namely:—

R. F. Montgomerie, Esq., PH.D., B.SC., F.R.C.V.S., Wellcome Research Laboratories, Langley Court, Beckenham, Kent; G. Atkinson, Esq., M.R.C.V.S., 4 Compton Grove, Nairn Road, Canford Cliffs, Dorset; L. G. Anderson, Esq., D.V.M., M.R.C.V.S., 49 Cambridge Street, Aylesbury, Bucks.; T. M. Mitchell, Esq., M.R.C.V.S., D.V.S.M., Briony, Meadowfield Road, Stocksfield-on-Tyne, Northumberland; and Professor R. Lovell, D.SC., PH.D., M.R.C.V.S., D.V.S.M., Royal Veterinary College, Camden Town, London, N.W.1.

The term of office of the 5 persons elected in the place of the 5 members retiring by rotation will be 4 years.

Nominations of persons for election to the Council must be submitted in writing on a form which may be obtained from the undersigned, and which must be returned to him duly completed on or before January 31st, 1961.

Each such nomination form must bear the signature of the proposer and seconder, who must not be present members of Council, and must be countersigned by the nominee.

W. G. R. OATES,
Registrar

Veterinary Surgeons Acts, 1881-1948
College House,
Wright's Lane,
Kensington, W.8.

NEW INVESTIGATION CENTRE

A new Veterinary Investigation Centre under the control of Mr. W. H. Parker has been established at Thirsk to serve the North Riding of Yorkshire. It is hoped that veterinary surgeons in general practice in the North Riding will utilise the facilities now available at Thirsk.

The address of the new Centre is:—

Ministry of Agriculture, Fisheries and Food, Veterinary Investigation Centre, West House, Station Road, Thirsk, Yorkshire. Telephone No. Thirsk 2065/6.

ROYAL AGRICULTURAL SOCIETY OF KENYA

Farmers' Days held at the Wellcome Research Laboratory (East Africa)

On November 22nd and 23rd, 1960, members of the R.A.S.K. were entertained by Messrs. Burroughs Wellcome and Company (E.A.) and Messrs. Cooper McDougall and Robertson (E.A.) Ltd., at the Wellcome Research Laboratory (E.A.) at Kabete near Nairobi.

The programme commenced at 11 a.m. on each day and included talks by Dr. S. F. Barnett, Senior Research Officer at the Kenya Veterinary Research Laboratories, and Dr. H. T. Carroll, Sheep Adviser to the United Nations Food and Agricultural Organisation. These talks, which covered such subjects as Nairobi Sheep disease, anaplasmosis and sheep husbandry, stimulated lively discussion and a great deal of interest was aroused. After a lunch served under the trees on the lawns in front of the laboratories, various demonstrations, including cattle spraying, sheep dosing and a field sheep *post-mortem* examination, were staged. Such was the interest of the many sheep farmers present, that the demonstrations were kept fully occupied until after 4 p.m., when tea was served.

Throughout the 2 days the laboratories were open to guests and demonstrations of the work proceeding were arranged. These included the methods being used for the screening of drugs against East Coast fever and anaplasmosis, and field trials of certain anthelmintics. The haematological and other techniques used in the research work of the laboratories were demonstrated. These attracted a good deal of interest and many visitors expressed appreciation of the work being done by the Wellcome Foundation Ltd. in this way, for the benefit of East African agriculture.

Subsequently, at a general meeting of The Royal Agricultural Society of Kenya, the President of that Society described the two days as the most successful events held by his organisation.

REPLIES TO ADVERTISEMENTS

It has come to our notice that concern is being caused to new graduates by the length of time which elapses when they have answered Appointments Vacant advertisements in THE VETERINARY RECORD, before they receive a reply. This may amount to several weeks, and in some cases no reply at all is forthcoming. Principals of practices are busy people, but we are sure they will understand that young men and women, just admitted to the profession, are anxious to get settled, and their applications for vacant appointments are matters of serious moment to them.

UNIVERSITY NEWS

London

Bachelor of Veterinary Medicine

HONOURS: Noakes, David Ernest.

PASS: Baker, Michael William; Barber, Kenneth William; Berry, Allan Philip; Bleby, John; Bourne, Frederick John; Callaghan, John Barrie; Cartwright, Jonathan Richard; Chambers, John Clive; Crossley, John Francis; Davies, Anthony Baumann; Doncaster, Robert Antony; Elliott, Donald Arthur; Esplen, William Brian; Evans, David Evan Gwenog; Evans, Keith; Featherstone, David John Lee; Foord, Henry Edward; Fraser, Hugh; Hails, Susan Patricia; Harris, John Douglas; Hill, Frederick William Graham; Hoad, Malcolm John; Hogger, David George Jones, Keith Rowland Morgan; Kally, Uziel; Lewis, Roger David; Manning, Valerie Alison; Moore, Bryan Michael; Pilling, Lionel John Christopher; Potter, William Richmond; Quest, John Averill; Redfern, David Michael Robertson, David Gilbert; Shaw, Richard Carter; Smith, Dennis Leonard; Sutton, Douglas Michael; Swainston, John Jerome Tinkler, Christopher John; Tribe, Howard Arthur; Unwin, David Dudley; Woode, Gerald Nottidge; and Woodward, Brian George.

The following Candidates have passed in Part II:—

Brewitt, James Michael; Hanson, Alan Arthur; Norris, Raymond John; and Willott, John Arthur.

ERRATA

We regret certain errors in recent issues of this journal. On page 1,178 (*Vet. Rec.*, December 17th, 1960) the second reference under Lovell, R. should have been to this author only and not to himself and a colleague. On page 1,198 of the same issue, under the General Discussion, line 7, the word andotoxin should have read exotoxin. In the following week's issue, on page 1,223, col. 2, line 2, the Royal (Dick) School of Veterinary Studies should have read "the Glasgow University Veterinary School."

Mr. HUGH FRASER

By the death of Mr. Hugh Fraser, which occurred on November 28th, 1960, a connexion of nearly 40 years with the agricultural community in the West of England was broken.

Mr. Fraser, who was aged 72, joined with the late Mr. G. H. Gibbings in practice at Tavistock in 1919 and retired from active practice in 1956.

He qualified in 1910 at the Royal (Dick) College, and held appointments first in Aberdeen and later at Liskeard and Bideford.

During the 1914-18 war he served in France with the R.A.V.C., and shortly after demobilisation, with the rank of Captain, settled in Tavistock.

Mr. Fraser held appointments as Veterinary Inspector to the Devon County Council and later to the Ministry of Agriculture. His particular professional interest was horses, and he was for some years hon. veterinary surgeon for the Lamerton and Spooners Hunts Point-to-Point Races.

He was a past President of Western Counties Veterinary Association.

Mr. R. R. Willing writes:—

On first acquaintance with Hugh Fraser one might not realise that a most courteous and generous disposition lay behind an apparently blunt exterior. He was respected by all who knew him and it was truly said of him that "his word was his bond."

It was my privilege to have known him for 25 years and I was grieved to learn of his death, but am grateful for the lasting memory of a true and valued friendship.

The following veterinary surgeons joined the relatives in tribute at a cremation ceremony in Plymouth on December 1st: Mr. P. Fisher, Mr. J. C. MacKellar, Mr. J. A. White, and Mr. R. R. Willing.

PERSONAL

Mr. H. B. Lomas, a well-known and popular figure at B.V.A. Congresses has been appointed to the Board of Directors of The Wellcome Foundation Limited, with special responsibility for sales and distribution. Mr. Lomas was in November, 1957, appointed General Sales Manager of the Veterinary Division, and on May 1st, 1960, became Assistant Sales Manager.

Mr. J. A. McCutcheon, who was stationed at Newport, Isle of Wight, retired from the Animal Health Division on December 31st, 1960.

Engagements

BURNSIDE—THOMSON.—The engagement is announced between Raymond Emerson Burnside, B.V.M.S., M.R.C.V.S., younger son of Rev. John W. Burnside and Mrs. Burnside, of Paisley, and Barbara, elder daughter of Mr. and Mrs. R. H. Thomson, of Paisley.

Births

MANKTELOW.—On December 30th, 1960, to Judy and Bill Manktelow, of Wallaceville Animal Research Station, New Zealand, a son, Guy William David, brother to Joanna.

THURGUR.—On December 7th, 1960, to Marie-Christine, wife of John Thurgur, B.V.M.S., M.R.C.V.S., 4 Collingwood Avenue, Rugby, a son, Stephen James.

B.V.A. EVIDENCE BEFORE OFFICIAL COMMITTEES

On Tuesday, January 10th, the President of the B.V.A. (Mr. Sydney Jennings), Mr. J. B. White, chairman of the Technical Development Committee, and Mr. A. J. Wright, chairman of the Farms Livestock Committee, presented a Memorandum on behalf of the B.V.A. to the Netherthorpe Committee on antibiotics in animal feeding-stuffs.

On Wednesday, January 11th, the President, Mr. Horace Bell, Mr. E. Brayley Reynolds, Mr. J. S. Cosgrove, Dr. A. C. Fraser, Mr. Wm. C. Miller, and the General Secretary gave oral evidence to the Duke of Norfolk's committee on the doping of race-horses.

FOOT-AND-MOUTH DISEASE

Restrictions on the movement and marketing of livestock in the Infected Area around Glasgow were withdrawn as from January 11th.

The 24 outbreaks in this area involved the slaughter of 3,510 cattle, 8,625 sheep, 423 pigs and 3 goats.

Restrictions were originally imposed around Glasgow on November 12th. Subsequently, the disease spread over a wide area of Central Scotland; the last outbreak occurred at Aberdour, Fifeshire on December 20th.

COMING EVENTS

January

20th, 21st and 22nd (Fri., Sat., and Sun.). Annual Conference of the Association of Veterinary Students in New Brighton, Wallasey.

25th (Wed.). B.S.A.V.A. METROPOLITAN REGION. Meeting at the Royal Society of Medicine, 1 Wimpole Street, London, W.1, 7 p.m.

STAFFS. AND DERBYS. VETERINARY CLUB. Meeting at the White Hart Hotel, Uttoxeter, 8 p.m.

26th (Thurs.). EASTERN COUNTIES VETERINARY SOCIETY. Special General Meeting in the Lecture Theatre of the School of Veterinary Medicine Madingley Road, Cambridge, 2.30 p.m.

27th (Fri.). NORTHERN IRELAND VETERINARY ASSOCIATION. Meeting at the Whitla Medical Institute, College Square North, Belfast, 7 p.m.

February

1st (Wed.). DUMFRIES AND GALLOWAY DIVISION. Meeting in the Queensberry Arms Hotel, Arran, 2.15 p.m.

4th (Sat.). B.S.A.V.A. SOUTHERN COUNTIES REGION. Meeting at the Royal Hotel, Winchester, 6.30 p.m.

7th (Tues.). 12th Anniversary Dinner-Dance of the University of Bristol Centaur Society at the Berkeley, Bristol.

9th (Thurs.). Annual Ball of the Royal (Dick) School of Veterinary Studies in the Assembly Rooms, George Street, Edinburgh, 9 p.m.

SOCIETY FOR THE STUDY OF ANIMAL BREEDING. 9th Annual General Meeting at the Royal Society of Medicine, 1 Wimpole Street, W.1, 10.30 a.m.

B.V.A. COMMITTEE & COUNCIL MEETINGS

Tuesday, January 17th, at 7, Mansfield Street

5 p.m. Finance Sub-committee.

Wednesday, January 18th, at 7, Mansfield Street

10.30 a.m. Farm Livestock Committee.

12 noon. Home Appointments Committee.

2.15 p.m. Small Animals Committee.

4 p.m. Parliamentary and Public Relations Committee.

Thursday, January 19th, at 7, Mansfield Street

10.30 a.m. Veterinary State Medicine Committee.

2.15 p.m. General Purposes and Finance Committee.

Friday, January 20th

10.15 a.m. Council Meeting, Connaught Rooms, Gt. Queen Street, London, W.C.2.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ayr. Ralstonhill Farm, Kilmarnock; Templeton Farm, Kilmarnock (Jan. 2).

Ches. Cock Inn Farm, Barton, Malpas (Jan. 7); Streetfield Farm, Vicars Cross, Chester (Jan. 9).

Cumberland. Mechi Farm, Blennerhasset, Aspatria, Carlisle (Jan. 2); Carleton Farm, Carleton, Carlisle (Jan. 7); Greengill Farm, Gilcrux, Aspatria, Carlisle (Jan. 9).

Denbighs. Lower Eyton Farm, Eyton, Wrexham (Jan. 4).

Lanarks. Millhill Farm, Pettinain (Jan. 4).

Leics. Dixon Lodge Cottage, Gipsy Lane, Humberstone (Jan. 9).

Norfolk Whitehouse Farm Banham, Norwich (Jan. 6); Ashtrees Farm, Attlebridge, Norwich (Jan. 7).

Staffs. Field Hall Farm, Field, Uttoxeter (Jan. 9).

Foot-and-Mouth Disease

Ches. Broughton Lodge, Threapwood, Malpas (Jan. 9).

Norfolk. Church Farm, Old Costessey, Norwich (Jan. 9).

Fowl Pest

Bedford. Coleshill Allotments, Bedford (Jan. 3).

Berks. Park View, Swallowfield, Reading; 188 Whitley Wood Road, Reading; Department of Agriculture, Lane End Farm, Shinfield, Reading (Jan. 3); Wheelers Farm, Swallowfield, Reading (Jan. 5); Hyde End Farm, Shinfield, Reading; Oaklands, Grovelands Road, Spencers Wood, Reading (Jan. 6); 170 Whitley Wood Road, Reading (Jan. 9).

Bucks. Stags Lodge Farm, Shenley Church End, Bletchley (Jan. 5).

Derbys. 156 High Lane East, West Hallam (Jan. 8).

Essex. Laurels, Panfield, Braintree; Dewgates, High Roding, Dunmow (Jan. 2); Mansion House Farm, Unit 3, Althorne, Chelmsford (Jan. 3); Highlands Poultry Farm, Rettenden, Chelmsford (Jan. 4); Shirley, Lower Park Road, Wickford; Lower Dunton Road, Horndon, Stanford Le Hope; The Whim, Hallbridge Road, South Woodham, Chelmsford (Jan. 5); Meadowside, Little Warley Brentwood; 35 Nipsells Chase, Mayland, Chelmsford; The Piggery, West Road, South Ockenden, Romford (Jan. 6); Grey House, Heybridge Basin, Maldon (Jan. 7);; Summer Hill Farm, Crayshill, Billericay (Jan. 8); Glenwood, Little Warley, Brentwood; The Willows, Rettendon Common, Chelmsford.

Hants. Firhill Farms, Hollywater, Bordon (Jan. 2).

Kent. Rigsby Poultry Farm, Longfield Avenue, New Barn, Lingfield (Jan. 8); Sylva, Ash Road, Hartley, Dartford (Jan. 9).

Lancs. Back o' th' Moss Farm, Unsworth, Bury (Jan. 3); Shaw Green Farm, Euxton, Chorley (Jan. 4); Deane Hall Lane, Euxton, Chorley (Jan. 6).

Norfolk. Street Farm, Irstead, Neatishead, Norwich; Smallholding, Eye Road, Hoxne, Diss; Town Street, Hickling, Norwich; Silver Street, Fleggburgh, Great Yarmouth; Bexley Farm, Irstead, Neatishead, Norwich; The Bungalow, Somerton Road, Martham, Great Yarmouth; Peasland Green, Elsing, Dereham; Broadfield, Hickling, Norwich (Jan. 2); Pear Tree Farm, Pulham Market, Diss; Four Winds, Beed Avenue, Taverham, Norwich; Brookdene, Drury Lane, Carbrooke, Thetford; Stubb Road, Hickling, Norwich: Bridge Farm, Hemsby, Great Yarmouth; King Georges Hall Cottage, Harleston; Six Oaks, Brick Lane Farm, Taverham, Norwich; The Gardens, St. Johns Lane, Sisland, Loddon, Norwich; Horse Shoes Public House, Branham Broom, Norwich (Jan. 3); Homestead, Bunwell Hill, Bunwell, Norwich; Church Road, Barrack, Reepham; The Allotments, Anson Road, Great Yarmouth (Jan. 4); Broadhaven Gardens, Station Road, Potter Heigham, Great Yarmouth; 12 Leman Grove, Loddon, Norwich; Whitehorse Farm, Tasburgh, Norwich; The Grove, Bungay Road, Loddon, Norwich; Drowick Hall, Wymondham; Green Piggery, Hurfreys Road, Great Yarmouth (Jan. 5); Highfield, Windmill Lane, Old Costessey, Norwich; Doles Farm, Bradwell, Great Yarmouth (Jan. 6); Old Home Cottage, Swafield, North Walsham; The Cottage, Sisland, Norwich (Jan. 7); Fifers Nurseries, Buxton Road, Spixworth; Walnut Tree Farm, Denton, Harleston; Deopham Road, Hingham, Norwich (Jan. 8); Whitehouse Farm, Town Street, Hevingham; Becketts Farm, Hempstead, Holt; Read Corner, Fleggburgh, Great Yarmouth; Whitehouse Farm, Rollesby, Great Yarmouth; The Laundry, Fleggburgh, Great Yarmouth; Broadlands Mushroom Farm, Martham, Great Yarmouth (Jan. 9).

Salop. Laburnham House, Prees Green, Whitchurch (Jan. 4).

Somerset. Highfields, Chilthorne, Domer, Yeovil (Jan. 2).

Suffolk. Hall Farm, Reydon, Southwold (Jan 2); Little Greenlands, Saxtead, Woodbridge (Jan. 4); Whitehouse, Saxtead, Woodbridge; Hall Farm, Reydon, Southwold (Jan. 6); Barnes Farm, Wenham Road, Copdock, Ipswich (Jan. 7); Poplar Farm, Mendlesham, Stowmarket; Whitmores Farm, Dennington, Woodbridge (Jan. 9).

Surrey. Newlands Corner, Riding Stables, Clandon, Guildford (Jan. 2); Kimberley Farm, Little Bookham, Leatherhead (Jan. 5).

Sussex. Barwon Heads, Camber, Rye; Grove Farm, Iden Rye (Jan. 5).

Wilts. Moormead Allotments, Wroughton, Swindon (Jan. 3).

Worcs. Richmond Cottage, Pound Green (Jan. 3); Chamberlyn Road, Bewdley (Jan. 4).

Yorks. Queen Mary's School, Duncombe Park, Helmsley (Jan. 3).

Swine Fever

Anglesey. Tyn Mur, Rhostrehwfa, Llangefni (Jan. 5).

Berks. Blakes Oak Farm, Lodge Hill, Sunningwell, Abingdon (Jan. 9).

Ches. Yew Tree Farm, Poulton, Pulford (Jan. 3); The Hough, Malpas (Jan. 5).

Devon. Steart Farm, Horns Cross, Bideford (Jan. 2); Mounstephen Farm, Halberton, Uffculme, Cullompton (Jan. 4); The Piggeries, Orchard Hill, Northam (Jan. 9).

Durham. Copley Bent, Butterknowle, Bishop Auckland; Coniscliffe Grange Farm, Staindrop Road, Darlington (Jan. 2).

Essex. The Piggeries, Coopers Lane, Great Leigh, Chelmsford (Jan. 5); Crumps Corner Farm, Little Canfield, Dunmow (Jan. 9).

Herefords. New House, Kimbolton, Leominster (Jan. 9).

Hunts. Windy Ridge, Mereside, Ramsey, Huntingdon (Jan. 3).

Lancs. Crown Point Piggeries, Briscoe Lane, Manchester, 10 (Jan. 2); Back Lane, Moss Side, Inskip, Preston (Jan. 5); North View Cottage, Gisburn, Clitheroe (Jan. 9).

Lincs. 4 Messingham Road, Scotter, Gainsborough (Jan. 2); Piggeries, Sluice Farm, Gunness, Scunthorpe (Jan. 9).

Notts. Piggeries, Rhymes Farm, Carlton Lane, Sutton-on-Trent (Jan. 4).

Salop. Whitehouse Farm, Hemford, Minsterley, Shrewsbury (Jan. 3).

Somerset. Crossmoor Farm, Cannington, Bridgwater (Jan. 2); Manor Farm, Downhead, Shepton Mallet (Jan. 5).

Surrey. Broadwater Farm, Weybridge (Jan. 4).

Sussex. Seedhill Farm, Broad Oak, Heathfield (Jan. 2).

Wilts. Shurnhold Farm, Melksham (Jan. 4).

Worcs. St. Peters Farm, Holloway, Droitwich (Jan. 2).

York. Craige Weil Cottage, 31 Back Lane, Moortop, New Farnley, Leeds, 12; Coffee House Buildings, Everingham (Jan. 3); Bungalow Farm, Woodhall Way, Beverley (Jan. 4); Carr Farm, Long Riston, Hull (Jan. 9).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

GENERAL

A Veterinary Centre

Sir,—Some weeks ago Mr. G. N. Henderson proposed the formation of a Veterinary Centre. This suggestion was surely one of the most progressive and far seeing that has appeared in THE VETERINARY RECORD for many years.

One or two letters supported Mr. Henderson; the majority of the profession remained silent. This silence, I suspect, is due to the fact that there is very little that one can add to this momentous plan.

I am sure, however, that we are all waiting to hear that this plan has been approved by the R.C.V.S. and B.V.A. The time is ready for a statement from these, our two ruling Associations. The B.V.A., I believe, has a Public Relations Officer and Secretary. The R.C.V.S. has its Registrar. Could we ask, Sir, through your columns if these gentlemen could give us a statement now?

Yours faithfully,
R. S. TOWNSON.

Kelperland,
Monkey Island Lane,
Bray,
Maidenhead.
January 6th, 1961.

Exchange of Students During Vacation

Sir,—The opportunity exists for British students to see practice abroad, for periods of one month, on an exchange basis. There are also many foreign students who would like to enjoy the advantages of such exchanges but we know very few practising veterinary surgeons who would be willing to take them.

I should be very grateful if any veterinary surgeon who would be willing to help in this way during the summer of 1961 would write to me giving details of the type of practice, dates convenient, and facilities offered.

Yours faithfully,
J. F. STATON.

Royal Veterinary College,
London, N.W.1.
January 10th, 1961.

B. Virus in Monkeys—A Warning

Sir,—At a meeting of the Central Veterinary Society on January 5th, 1961, a "Forum on Exotic Pets" was held and one of the subjects raised was the risk to human life of handling monkeys affected with a virus of the Herpes type known as B. virus.

Whilst the existence of this and other monkey-borne diseases is well known to those working with monkeys in laboratories, the meeting felt that many veterinary surgeons in practice may not be aware of this possible danger when handling their occasional primate patients especially newly imported Rhesus and Cynomolgus monkeys.

The disease in monkeys may cause lesions on the face, lips, mouth and even the body but it is possible that asymptomatic carrier states may occur. In man the disease is rare but has virtually a 100 per cent. mortality rate.

Yours faithfully,
E. C. APPLEBY,
Hon. Secretary,
Central Veterinary Society.

Dept. of Pathology,
Royal Veterinary College,
Royal College Street,
Camden Town,
London, N.W.1.
January 6th, 1961.

Veterinary Education

Sir,—Such experience as I had as a student provokes me to offer the following contribution to the above-named correspondence.

Firstly: excessive note-taking should be avoided; it does not, except by inordinate labour, promote thought or knowledge.

Secondly: where possible teachers should inform students what subject will be discussed at the following "lecture"; the students should then read it up and be prepared to answer and ask questions, and to discuss during the whole of the period of the "lecture." This method promotes thought, encourages clarity of expression and acquirement of knowledge.

These principles are not my own; they are gleanings from my best teachers and I believe that Socrates had much the same ideas. When they are followed they have the advantage that, however drowsy the day, neither teacher nor students doze, perhaps the latter gentlemen may become a little fearful, but at least their lives are increasingly full of excitement, and they become filled with a curiosity for, and an acquirement of, knowledge.

The method also allows an added amount of time for wider reading than that which is afforded by only digesting the assigned textbooks, and the student never suffers from writer's cramp of either hand or head.

Yours faithfully,
EDWARD F. PECK.

The F.A.O. Mission,
Box 1039,
Addis Ababa,
Ethiopia.
January 5th, 1960.

Worse than the Disease

Sir,—It was with great interest that I read of the valuable individual research carried out by Mr. George Tullis (*Veterinary Record*, December 31st, 1960).

I, myself, expend limitless energy in trying to avoid work of any kind. I feel now that this energy could be channelled into something worthwhile. I therefore offer my services to Mr. Tullis should he wish to form a research team to help solve this ever recurring problem.

Unfortunately, as with all research, funds are not readily available and, being a comparatively poor profession, it would be impossible to undertake this important work without some form of grant or subsidy.

As a small contribution to Mr. Tullis's work I would suggest three promising curative treatments:—

(1) Climb Ben Nevis.
(2) Swim the channel.
(3) Ride round Aintree.

The results are considerably improved if all three are undertaken in the one day.

Finally, I would like to warn intending volunteers of the very real danger to health and morals which this investigation might entail. However, as with all research workers, personal danger and suffering, I am sure, would be set at nought when considering the ultimate benefit to mankind.

I look forward, with confidence, to the day when the "wish I was dead" syndrome (Tullis, *Vet. Rec.*, **72**. 1,252) is no more.

Yours faithfully,
A. L. BARTHOLOMEW.

Victoria Road,
Darlington.
January 9th, 1961.

Diseases of the Bovine Foot

Sir,—The symposium on "Diseases of the Bovine Foot" published in THE VETERINARY RECORD, Vol. 72, No. 52, prompts us to mention the success we have achieved in treating deep suppurating lesions of the bovine foot.

Until a few years ago, amputation of the digit was our normal procedure in these cases, when chemotherapeutic methods failed.

It was decided to treat one cow affected with deep necrosis of the digit, with sinus formation, by enclosing the entire foot and fetlock joint in plaster of Paris. The plaster, thickly applied, was built up under the sound claw to relieve pressure on the affected digit, and this alone appeared to give considerable relief almost immediately.

Within a few days the cow's appetite and general condition were considerably improved. When the plaster was removed a month later, all the sinuses had healed, and the cow walked (and ran) normally. Since then we have used this treatment in all similar cases and have not needed to resort to amputation.

Other cases in which the closed plaster method of treatment has proved successful include compound fractures of limb bones, even in sheep and young cattle; open joints and severed tendons. It also provided the answer, when, after the removal of a large tumour from the fore-limb of a dog, it was found there was insufficient skin to effect a surgical repair.

Yours faithfully,
C. J. BAXTER,
H. FORSYTH.

Lechlade,
Glos.
December 28th, 1961.

The Future of Animal Husbandry in Overseas Territories

Sir,—In his article under this title, published in THE VETERINARY RECORD of January 7th, Mr. Miller calls for comments from both past and present members of the overseas services and this must be my excuse for putting forward once more some of my thoughts on this subject.

In my view the case for placing the overall direction of livestock improvement work, which is what animal husbandry means, under the unified control of a veterinarian is an overwhelming one in the tropics. How can one expect livestock to prosper if their requirements are attended to by two or more departments, which are often at loggerheads? How can one expect a veterinarian to carry out his duties efficiently if he is debarred from entering the field of nutrition in a country where the provision of a maintenance ration is often as important as finding a remedy for a contagious disease; or from giving advice on breeding problems in a country where the subject of controlled breeding is little understood and rarely practised? How can one expect an agriculturist to find time to give detailed study to the subject of livestock improvement when he has so many other irons to keep hot in the fire? And to the imposing list of such subjects given by Mr. Miller I would add agricultural engineering, which to-day is a major concern with all agriculturists!

I often think that we, as a profession are much to blame for the present unsatisfactory position of the veterinarian in the sphere of animal husbandry in the tropics. In the past quite an appreciable percentage of veterinary graduates have been slow to put forward any claim to expertise in this subject and this, I believe, has been due to two reasons. Although, as Mr. Miller points out, veterinary graduates are the best basic material from which to recruit animal husbandmen, the small amount of attention paid to this subject in our teaching institutions in the past has resulted in lack of confidence in our graduates. Again, the narrowness of the system of education in the past has provided graduates with an excuse for adopting the "closed shop" attitude and confining their attention strictly to purely veterinary problems, into which no non-veterinarian may enter.

We cannot have it both ways. If we wish to adopt the "closed shop" attitude then we must expect to see much interesting and rewarding work done by others; whereas if we broaden our horizon and open our doors to anyone interested in livestock improvement there will be competition in due course from non-veterinarians for some of the higher posts, including the ultimate head. May the best man win, but for the sake of smooth running may he be a veterinarian!

One last point on the question of nomenclature. I once heard a senior civilian, who afterwards became Governor of a province, say that he could do so much more for the veterinary staff, if only they would change their name. There seems to be a deep-rooted feeling amongst administrators that their veterinary department should not be allowed to do anything more than try to control disease. Maybe that feeling is passing as time goes on, but in India we took the hint some years ago and most of the provincial veterinary departments are now known as departments of animal husbandry. These departments deal with all questions relating to animals, and the introduction of this arrangement acted like magic. From a state of confusion which previously existed in the minds of administrators and the general public, as to where to go for advice on an animal subject, the picture suddenly became clear and these new departments began to function like well-oiled machines.

The effect on the veterinary staffs has also been very marked. No longer does the veterinary graduate feel that he is a member of the Cinderella of the services, but that he is now in the position of being able to deal with all aspects of a very important subject, the furtherance of which, in many areas in the tropics, will do more than anything else to raise the standard of living amongst the human population.

In case this letter is read by any of my agricultural friends, I should like to add that I hope nothing that I have said will detract from the spirit of good fellowship which has always existed between agricultural and veterinary staffs, at least in India. In making these proposals for a change in organisation it has always been my desire that these changes, if introduced, would not interfere with, but would rather help, the agriculturist to obtain the success he is striving to get in the rural economy of the country in which he is working.

Yours faithfully,
FRANK WARE.

Springfield,
Highclere,
Nr. Newbury,
Berks.
January 9th, 1961.

SCIENTIFIC

A Mild Form of East Coast Fever

Sir,—All previously described strains of *Theileria parva* have been shown to be highly pathogenic for cattle of exotic breeds. The case mortality rate for our laboratory strain, when high-grade cattle were infected, was 96 per cent., and in South Africa the mortality rate in calves was more than 95 per cent. In exceptional cases it may be as low as 78 per cent. (Neitz, 1957). This high case mortality rate has led veterinarians and farmers in Africa to believe any treatment that is followed by recovery to be curative.

This note records the isolation and maintenance of a strain of *T. parva* which caused a mortality rate of only 23 per cent. in high grade stock. Since the parasite was found on the farm of Mr. D. G. Icely, near Rumuruti, for convenience we refer to it as *T. parva* (Icely).

During attempts to determine the vector of *T. lawrencei* cattle were employed as bait animals for the collection of adult *Rhipicephalus appendiculatus*, the proved vector of *T. lawrencei* in South Africa. The cattle were exposed in a highland forest area which had been free of cattle for at least 5 years. Ticks were collected from the bait cattle before they had become firmly attached to the animals and brought back to the laboratory where they were allowed to feed on grade cattle. This resulted in the isolation of a parasite which has now been maintained through 7 cattle/tick passages. Fifty-seven cattle have so far been infected with *T. parva* (Icely) and only 13 of these have died. The mortality rate was related to the number of ticks used to transmit the organism; the mortality rate when 3 or 5 ticks were used was 0 per cent. (14 cattle); when 10 or 20 ticks were used it was 21 per cent. (33 cattle) and when 30 to 100 ticks were used it was 60 per cent. (10 cattle). Thirty-four of the cattle which recovered were challenged with highly virulent strains of *T. parva* and all were solidly immune. Further reasons for identifying this parasite as a strain of *T. parva* are as follows:—

1. Macroschizonts, microschizonts and intra-erythrocytic piroplasms were produced.

2. The parasites were morphologically identical with *T. parva* (Muguga) and *T. parva* (South Africa). Five hundred schizonts from a recovering case of *T. parva* (Icely) were found to have an average size of 4.3 μ and the average number of nuclei, in 5,850 schizonts, 7.97. The comparable figures for *T. parva* (Muguga and South Africa) were 4.4μ and 8.09 nuclei per schizont (Barnett *et al.*, 1961).

It is important for veterinarians in East Africa to

know that such mild strains may exist, particularly when assessing the value of field treatments.

A full description of this strain will be published later.

Yours faithfully,
S. F. BARNETT*,
D. W. BROCKLESBY.

East African Veterinary Research Organisation,
Muguga,
P.O. Box 32,
Kikuyu,
Kenya.

December 30th, 1960.

References

BARNETT, S. F., BROCKLESBY, D. W., & VIDLER, B. O. (1961). *Res. vet. Sci.* **2**. 11.

NEITZ, W. O. (1957). *Onderstepoort J. vet. Sci.* **27**. 275.

* Present address: Veterinary Research Laboratories, P.O. Kabete, Kenya.

An Unusual Case of Diabetes Mellitus in a Bitch

Sir,—This letter describes the biochemical findings and *post-mortem* lesions in a case of diabetes mellitus in a 7-year-old Golden Labrador bitch.

When the bitch was first seen her urine was found to contain ketone bodies and more than 2 per cent. reducing substances. There was a high concentration of glucose in the blood (240 mg. per 100 ml).

Tests for pancreatic function were carried out in an attempt to demonstrate any gross deficiency of this organ. Diastase is secreted by the pancreas into the intestine. It is then absorbed into the blood and excreted *via* the kidney. An assay of this enzyme in the urine (by measuring its ability at varying dilution to hydrolyse a standard starch solution) gave a value of 4 Wohlgemuth (diastase) units, compared with the normal range of 6 to 30 units. Despite this low value for diastase it appeared that lipase and trypsin were being secreted in adequate amounts, since the faeces were of normal consistency and microscopical examination showed normal digestion of muscle fibre.

The animal was stabilised on intramuscular zinc insulin, but the bilateral cataract which had been noted during the clinical examination had become much more marked in the meantime, and the owner decided to have the bitch destroyed.

Post-mortem examination of the pancreas showed marked abnormality. There was only a small portion of macroscopically normal tissue, situated anteriorly. This was drained by the anterior pancreatic duct opening into the duodenum with the bile duct at the major papilla (sphincter of Oddi). The middle part of the pancreas was thickened and appeared fibrotic, while the posterior part was small and atrophied. Both these parts of the pancreas were drained by the posterior pancreatic duct, opening into the duodenum at the minor papilla. This duct was obstructed just before it entered the duodenum by a calcified thickening in its wall which occluded the lumen. No other abnormalities were detected.

The histological report showed that the anterior pancreatic tissue was normal. The central part of the pancreas showed much replacement fibrosis and some fatty infiltration. Islets of Langerhans were difficult to find, and in some places were replaced by fibrotic tissue.

The present case makes an interesting comparison with that described by Singleton and Rhodes (1957), with the exception that in their case the anterior pancreatic duct, together with the bile duct, was obstructed near the major papilla by a small abscess. These authors suggested that the obstruction caused bile to pass up the anterior pancreatic duct and produced an acute haemorrhagic pancreatitis. In the present case it was the posterior pancreatic duct which was obstructed, and as shown by Stein, Powers and Browne (1956), this causes an interstitial pancreatitis. A chronic pancreatitis of this type would give rise to fibrosis, as was found in the middle part of the pancreas at *post-mortem* examination. The exact cause of the obstruction in the duct wall was not determined, although it seems possible that, as in the case described by Singleton and Rhodes (1957), it had started as a small abscess.

Pancreatic tissue anteriorly appeared normal, where it was drained by the anterior pancreatic duct, and it was presumably *via* this route that pancreatic digestive enzymes reached the intestine, although in smaller amounts than normal (as shown by the test for diastase activity).

I would like to thank colleagues at the School of Veterinary Medicine, Cambridge, for their advice and help.

Yours faithfully,
D. B. ROSS.

The Animal Health Trust,
Farm Livestock Research Centre,
Stock,
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DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN
Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
1st to 15th December, 1960		15	—	31	218	—	47
Corresponding period in	1959	8	—	24	375	—	62
	1958	13	—	—	38	—	78
	1957	8	—	30	62	—	36
1st January to 15th December, 1960		200	1	265	2,035	—	1,178
Corresponding period in	1959	245	2	37	1,752	—	1,258
	1958	156	5	116	705	—	1,224
	1957	307	8	165	959	—	921